PEAR

The Journal of HEREDITY

DOMINANCE NO PROBLEM HERE
(SEE PAGE 251)

A "Kinky-Tail" Gene in Cattle
Identical Twins with Tuberculosis
Genetics of Hernia in the Albino Rat
The Geneticist—is he Nazi or Bolshevik?
A Linkage-Similarity in three Rodent Genera
The Eugenist—Guide or Racial Weather Prophet?

VOLUME 27 JULY, 1936 NUMBER 7

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their application to the improvement of plants, animals, and human racial stocks

DAVID FAIRCHILD, President Vice-President, W. E. CASTLE Secretary, C. E. LEIGHTY

Treasurer, J. H. KEMPTON

Legal Advisor, STANTON C. PEELLE

EDITORIAL BOARD

Managing Editor, ROBERT C. COOK

M. DEMEREC R. R. GRAVES L. C. DUNN

H. J. MULLER GEORGE M. DARROW PAUL POPENOE

Members of the Council are ex-officio members of the Executive Board

COUNCIL

W. E. CASTLE, Professor of Genetics, Harvard University, and Research Associate of the Carnegie Institution of Washington.

LEON J. COLE, Professor of Genetics, University of Wisconsin.

G. N. COLLINS, Botanist, U. S. Department of Agriculture, Washington, D. C.

O. F. COOK, Bionomist in Charge of Breeding Investigations, U. S. Department of Agriculture.

DAVID FAIRCHILD, Agricultural Explorer, U. S. Department of Agriculture.

HARRY V. HARLAN, Senior Agronomist in Charge of Barley Investigations, U. S. Department of Agriculture.

SAMUEL J. HOLMES, Professor of Zoology, University of California, Berkeley, California.

C. E. LEIGHTY, Agronomist in Charge of Dry Land Agriculture, U. S. Department of Agriculture.

N. D. C. LEWIS, Chief Medical Officer, St. Elizabeth's Hospital, Washington, D. C.

J. H. KEMPTON, Bureau of Plant Industry, U. S. Department of Agriculture.

E. W. SHEETS, Chief of Animal Husbandry Division, Mississippi Agr. Expt. Sta., State College, Miss.

SEWALL WRIGHT, Professor of Zoology, University of Chicago.

ADVISORY COMMITTEE

W. S. ANDERSON, Genetics Department, University of Kentucky, Lexington, Kentucky.

E. B. BABCOCK, Genetics Department, University of California, Berkeley, California.

E. A. BESSEY, Professor of Botany, Michigan State College, East Lansing, Michigan.

A. F. BLAKESLEE, Plant Genetist, Carnegie Institution, Cold Spring Harbor, New York.

J. K. BREITENBECHER, McGill University, Montreal, Canada.

W. E. BRYAN, Professor of Plant Breeding, University of Arizona, Tucson, Arizona.

C. B. DAVENPORT, Carnegie Institution of Washington, Cold Spring Harbor, L. I., New York.

J. A. DETLEFSEN, Professor of Genetics, Wistar Institute, Philadelphia, Pennsylvania.

RUSSELL W. DUCK, Livestock Editor, *The Rural New Yorker*, Syracuse, N. Y.

E. M. EAST, Professor of Plant Genetics, Bussey Institute of Applied Biology, Boston, Mass.

R. A. EMERSON, Professor of Plant Breeding, Cornell University, Ithaca, New York.

C. P. GILLETTE, President, The Genetic Foundation, Fort Collins, Colorado.

C. M. GOETHE, Sacramento, California.

HARRISON R. HUNT, Professor of Zoology, Michigan State College, East Lansing.

H. S. JENNINGS, Professor of Zoology, Johns Hopkins University, Baltimore, Maryland.

D. F. JONES, Plant Breeder, Connecticut Agricultural Experiment Station, New Haven, Conn.

M. A. JULL, Senior Poultry Husbandman, U. S. Department of Agriculture, Washington, D. C.

F. B. LINFIELD, Director, Montana Experiment Station, Bozeman, Montana.

C. C. LITTLE, Director, Roscoe B. Jackson Memorial Laboratory, Bar Harbor, Maine.

JAY L. LUSH, Professor in Animal Husbandry, Iowa State College, Ames, Iowa.

H. H. Newman, Professor of Zoology, University of Chicago, Chicago, Ill.

T. S. PALMER, Expert in Game Conservation, U. S. Dept. of Agriculture, Washington, D. C.

JOHN H. PARKER, Professor of Agronomy, Kansas State College, Manhattan, Kansas.

JOHN W. SCOTT, Professor of Zoology, University of Wyoming, Laramie, Wyoming.

A. D. SHAMEL, Physiologist, U. S. Department of Agriculture, Riverside, California.

GEORGE H. SHULL, Professor of Biology, Princeton University, Princeton, New Jersey.

EDMUND W. SINNOTT, Professor of Botany, Barnard College, Columbia University, New York City. (Representative of A. G. A. on Council of Amer. Assoc. for the Advancement of Science.)

W. W. SMITH, Professor of Animal Husbandry, Purdue University, Lafayette, Indiana.

CHARLES R. STOCKARD, Professor of Anatomy, Cornell University, New York City.

A. B. STOUT, Director of Laboratories, New York Botanical Gardens, New York City.

W. T. SWINGLE, Plant Physiologist in Charge of Breeding Investigations, U. S. Department of Agriculture, Washington, D. C.

W. H. TOMHAVE, American Aberdeen-Angus Breeders' Association, Chicago, Ill.

H. E. WALTER, Professor of Biology, Brown University, Providence, Rhode Island.

H. J. WEBBER, Prof. Subtropical Hort., Citrus Experiment Station, Riverside, California.

E. N. WENTWORTH, Armour's Bureau of Agr'l Research and Economics, Chicago, Illinois.

P. W. WHITING, Station for Experimental Evolution, Cold Spring Harbor, Long Island, N. Y.

The JOURNAL OF HEREDITY is published monthly by the AMERICAN GENETIC ASSOCIATION at 1918-32 Harford Avenue, Baltimore, Md., and Victor Building, Washington, D. C. Address all communications regarding editorial matters to Room 308, Victor Building, Washington, D. C. Communications regarding the business management of The JOURNAL OF HEREDITY may be addressed to the publication office, 1918-32 Harford Avenue, Baltimore, Md., or to the Victor Building, Washington, D. C. Entered as second-class matter February 24, 1923, at the postoffice at Baltimore, Maryland. Acceptance for mailing at the special rate of postage provided for in section 1103, Act of October 3, 1917, authorized December 20, 1918.

Contents copyrighted 1936 by the American Genetic Association. Unauthorized reproduction of material from THE JOURNAL not permitted. The title "Journal of Heredity" is registered as a trade-mark in the U. S. Patent Office.

$3.50 a Year. 35c the Copy. Prices of back numbers on application.

The

Journal of Heredity

Vol. XXVII JULY, 1936 No. 7

ORIGINAL ARTICLES

THE STRUCTURE OF THE SEX-CHROMOSOMES OF NEUROSPORA CRASSA
By Carl C. Lindegren 251

LINKAGE OF PINK-EYE AND ALBINISM IN THE DEER-MOUSE
By Frank H. Clark 259

A CHALLENGE TO THE EUGENIST
By J. P. Scott 261

A PAIR OF TUBERCULAR TWINS
By D. R. Atzenhoefer and D. Cecil Rife 265

THE INHERITANCE OF SCREW TAIL IN CATTLE
By Bradford Knapp, Jr., M. W. Emmel, and W. F. Ward 269

INHERITANCE OF UMBILICAL HERNIA IN RATS
By L. A. Moore and P. J. Schaible 273

Notes and Book Reviews

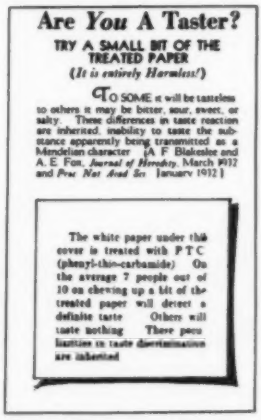
$1,000.00 FOR A "POPULAR SCIENCE" MANUSCRIPT 260

GENETICS AND POLITICS 267

MARRIAGE IN THE COLLEGE CURRICULUM 268

MORE ABOUT THE DOUBLE NASTURTIUM PATENT 271

THE JEW IN SCIENCE (Rev. of L. GERSHENFELD, *The Jew in Science*) 280

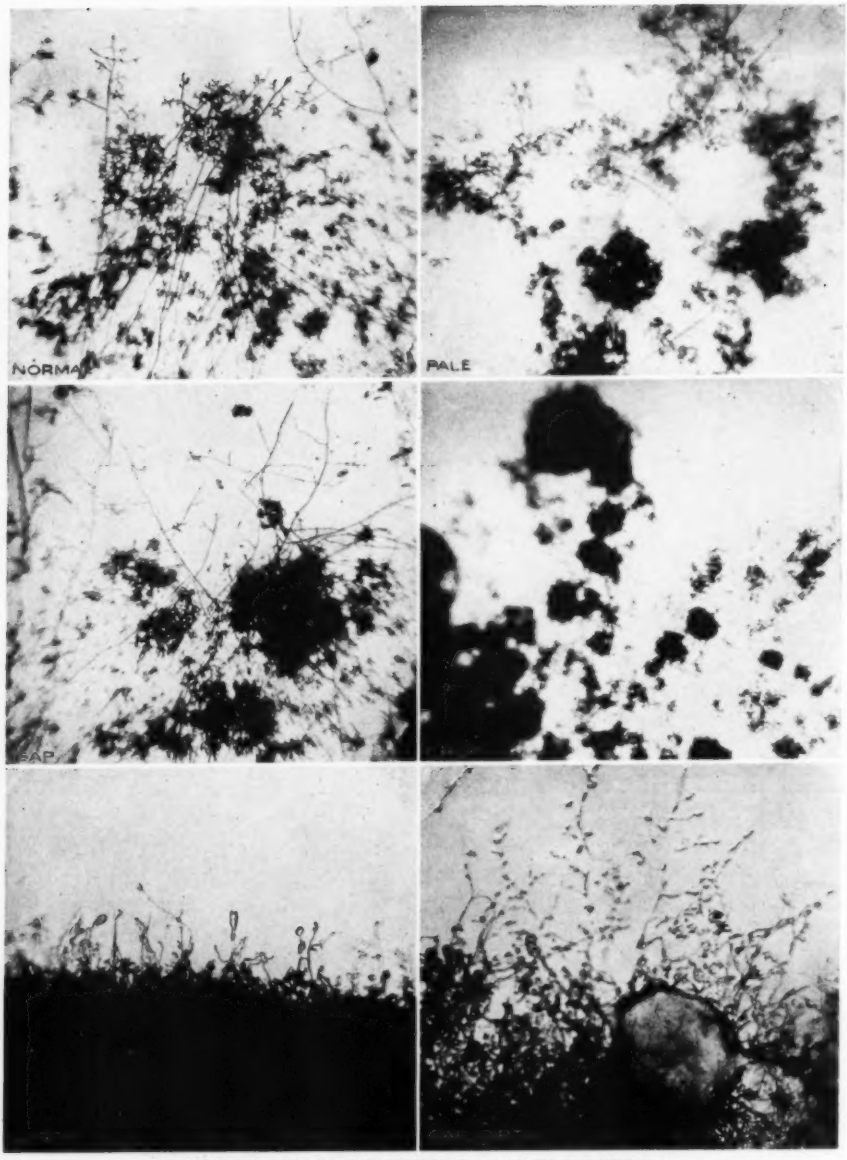

Are You A Taster?
TRY A SMALL BIT OF THE TREATED PAPER
(It is entirely Harmless!)
TO SOME it will be tasteless to others it may be bitter, sour, sweet, or salty. These differences in taste reaction are inherited. inability to taste the substance apparently being transmitted as a Mendelian character. [A. F. Blakeslee and A. E. Fox, Journal of Heredity, March 1932 and Proc. Nat. Acad. Sci. January 1932.]
The white paper under this cover is treated with P T C (phenyl-thio-carbamide) On the average 7 people out of 10 on chewing up a bit of the treated paper will detect a definite taste Others will taste nothing There peculiarities in taste discrimination are inherited

LEAFLETS TO DEMONSTRATE HEREDITY

Last year the "Taster test" served as an introduction to human genetic differences in many classrooms. The test is so easily made with the treated paper, and arouses such great interest that it appears to have a definite place in group demonstrations of such differences. These leaflets, size 3⅜" by 5½", are available in quantities for those who wish to use them for classroom or lecture purposes. On the back is printed an outline 4-generation pedigree chart. Sample on request. Prices: 10 for 25c; 50 for $1.00.

AMERICAN GENETIC ASSOCIATION

308 VICTOR BUILDING WASHINGTON, D. C.



MENDELIAN CHARACTERS IN NEUROSPORA

Frontispiece

Photographs of mycelia of normal *Neurospora crassa* compared with four variants each produced by single gene differences and by a combination of two of these genes (*gap-dirty*). (The photographs were made by placing the culture tubes on the stage of a microscope and photographing through the glass, magnification approximately 20 diameters.) These genes are all located in the sex-chromosome (see Figure 4), and their linear arrangement is here discussed Since the mycelium is a haploid structure, it is possible in the fungi to study genic relations by inspection without recourse to breeding tests made necessary by the diploid nature of higher plants and animals. The fungus geneticist is spared the problems and the complexities introduced by dominance and recessiveness, but the existence of four sexes in some forms is a compensating complication.

THE STRUCTURE OF THE SEX-CHROMOSOMES OF NEUROSPORA CRASSA

Suggested by Genetical Analysis

CARL C. LINDEGREN
University of Southern California, Los Angeles

THE heterothallic ascomycete, *Neurospora crassa,* produces the diploid zygote nucleus in a long narrow sac, the ascus. This nucleus undergoes reduction and four haploid nuclei are produced which then divide equationally to produce eight haploid nuclei in each sac. The spindles in the ascus do not overlap, with the result that first- and second-division reduction can be distinguished by the arrangement of the spores. Each haploid spore can be grown in pure culture to produce a thallus. These thalli may be either of (+) or (—) sex, and oppositely sexed haploid thalli can be mated with each other and the progeny subjected to genetical analysis. The genetical constitution of each thallus can be determined directly by inspection. The classification of progeny is simplified by the haploid nature of the thallus. The difficulties imposed by recessiveness and dominance in diploid forms are not encountered.

Asexual reproduction is effected by asexual spores (conidia) which are borne on conidiophores, extensions of the vegetative fungal hyphae (Figure 1). A single conidium planted alone produces a mycelium (fungus mat) bearing conidiophores and conidia. Mycelia may be either of (+) or (—) sex. No visible criterion exists for distinguishing them. They are identified by growing them in the same test tube with a standard (arbitrarily chosen) tester strain. If the *tester* strain is (+) and the *tested* strain (—) (or vice versa) perithecia result. These are large (250 microns), carbonaceous, flask-shaped bodies with thick brittle walls. When a perithecium is crushed between two glass slides, a cluster of asci is expelled. In Figure 2, such a cluster is shown. The ascospores when ripe are black; the lighter ascospores are unripe. Each ascospore is either of (+) or (—) sex. When a single ascospore is planted alone in a test tube, it produces a mycelium bearing conidiophores and conidia indistinguishable from the mycelium produced by growing a single (+) or (—) conidium alone. In closely related forms the existence of four distinct sexes instead of the "conventional" two shows that the criteria for distinguishing sex need more precise definition. An attempt will be made in the discussion to establish a fundamental basis for defining sex. Sex organs are not satisfactory criteria because many organisms like the Hymenomycetes have no sex organs but are capable of producing zygotes. Since the production of a zygote is patently a sexual act, it was chosen as the fundamental criterion.

A number of distinct characters have been isolated in this species and have been shown by appropriate crosses to be Mendelian in their inheritance. In this paper it is proposed to discuss the structure of the sex chromosome as it is revealed in the linkage relations of three known genes which have been located in the same chromosome as the plus-minus pair of sex-differentiating genes, or groups of genes and the spindle-fiber attachment.

Mycelial Characters

The different genotypes can be distinguished by microscopic examination. In Frontispiece are shown photomicrographs of the mycelia taken by placing the culture tube on the microscope stage and photographing directly through the glass. Colored natural size photographs of all these forms have been previously

published by the writer.[9] The magnification of the photomicrographs is not sufficient to show individual conidia. The *pale* mutant is characterized by a considerable reduction of the length of the condiophore and a consequent clumping of the conidial clusters, as well as by a difference in color. In the case of the *crisp* mutant, this reduction in length of the conidiophore is much more conspicuous and all the conidia are in tight clusters. There is also a color difference. In the *gap* mutant there is at first a considerable mycelial growth with few conidia. As the culture grows older, conidia are produced at the upper part of the tube in large, loose clusters.

In the *dirty* mutant, no true monilioid conidia are produced. An abundance of yellowish exudate and a few misshapen conidia are produced in a dense mass of tightly interwoven hyphae. The exudate characteristic of *dirty* is also found in the multiple mutants carrying the *dirty* gene, as the photomicrograph of the *gap-dirty* mutant shows.

Breeding Experiments

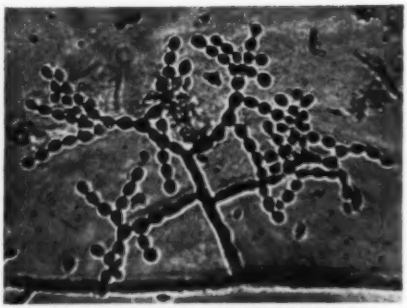
In two earlier papers the writer[7,9] presented data concerning loci in the sex-chromosome of *Neurospora crassa*. *Crisp* and *pale* were found to be linked[9] but were not located exactly. Eighty-eight asci were analyzed from the cross (—), *pale, dirty* by (+), *crisp* $\left(\frac{-\ c\ P\ D}{+\ C\ p\ d}\right)$ The number of original combination and recombination progeny are shown in the following tabulation.* Since every character was not classified in every case, the totals for the different linkage calculations are not the same.

The percentage of recombination for a locus and the spindle-fiber-attachment (s.f.a.) can be determined by dividing the percentage of second-division segregation of a pair of alleles by 2 (Lindegren[7]). The percentages of recombination† for the four loci and the s.f.a. were calculated on this basis.

The measurements resulting from this analysis are shown in the map (Figure 4). The ratio:

$$\frac{\text{recombinations}}{\text{original combinations plus recombinations}}$$

approaches .50 as a limiting value. When the distance between two pairs



CONIDIA AND CONIDIOPHORES

Figure 1

The ascospore when planted gives rise to a mycelium, a mat of fungal threads, in the agar substrate. From these threads aerial branches (conidiophores) are sent up which bear the conidia (asexual spores). This photograph shows the structural detail of the conidial mass depicted in the Frontispiece. All the nuclei in a given mycelium grown from a single ascospore (and in the resulting conidia) are genotypically identical and all of the same sex [(+) or (—)] as the original ascospore. The two sexes can be distinguished only by testing them with a standard tester strain to see whether sexual bodies (perithecia and ascospores) are produced. If none appear the strain being tested is of the same sex as the tested strain. If perithecia are produced the sex of the "unknown" is opposite to that of the tested strain. Each single conidium when planted on an agar substrate will produce in turn a mycelium identical to that from which it arose. Magnified about 250 diameters.

*TABLE I. Recombination between four loci in the Sex-Chromosome

Original combination		Recombination		Recombination %
+C, —c	548	—C, +c	92	14.4
—P, +p	510	+P, —p	126	19.8
—D, +d	392	+D, —d	248	38.7
Cp, cP	640	CP, cp	64	9.0
Cd, cD	444	CD, cd	260	36.7
PD, pd	448	Pd, pD	256	35.4

†TABLE II. First- and Second-Division Asci

Alleles	First-Division Segregation	Second-Division Segregation	Distance from s. f. a.
+/—	67	13	8.1
C/c	74	14	9.4
P/p	65	23	13.0
D/d	32	56	31.8

SEXUAL PHASE OF THE NEUROSPORA REPRODUCTIVE CYCLE

Figure 2

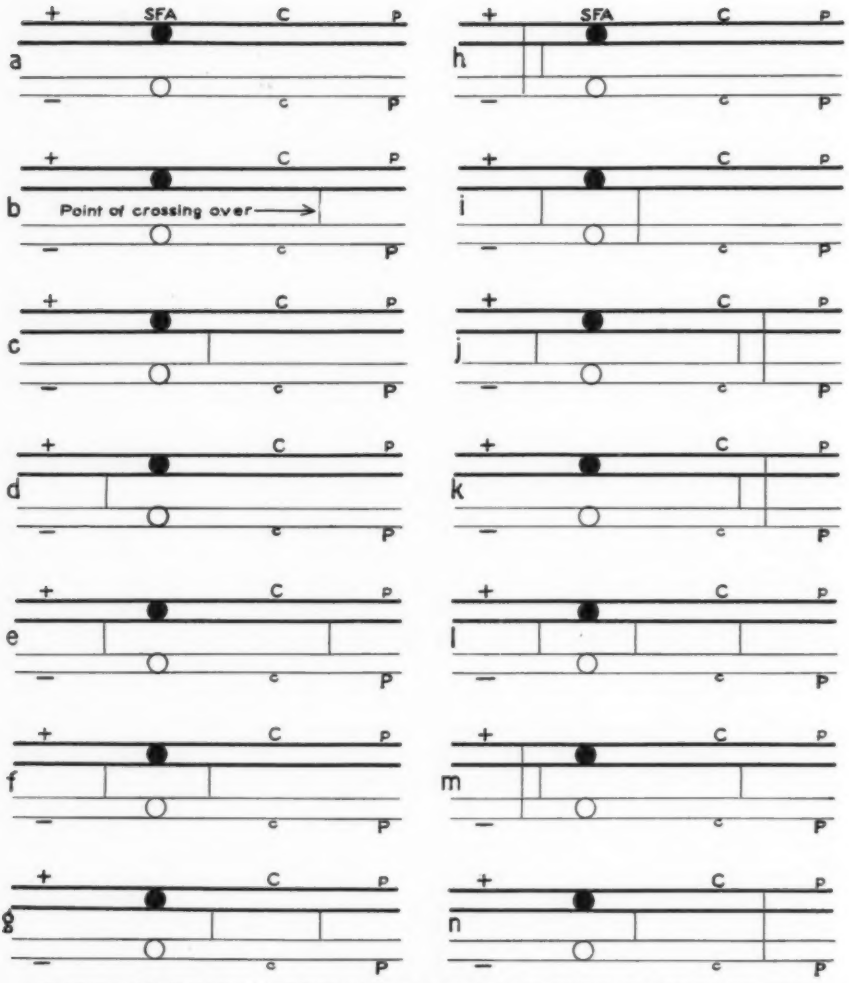
A portion of a cluster of asci (sacs) each containing ascospores (sexual progeny). These asci are formed in a perithecium which results from growing (+) and (−) mycelia together. At (or before) the initiation of the perithecium there is a fusion of the cytoplasm (plasmogamy) by the anastomosis of fungal threads (hyphae) from the (+) and (−) mycelia. This brings together a pair of (+) and (−) nuclei in a mixture of (+) and (−) cytoplasm. The nuclei do not immediately fuse (karyogamy is frequently delayed in fungi) but line up side by side and divide conjugately until a large number of pairs have been produced. By a special type of division, each pair is isolated in a young ascus. In each of these sacs a pair of associated nuclei (which has been cut off from the rest of the cytoplasm by a cell wall) fuse to produce a zygote nucleus. The diploid nucleus immediately undergoes reduction producing four nuclei. Each of these divides again and eight nuclei are produced in each ascus. An ascospore wall is produced about each of these eight nuclei. In the center of the photograph, one long straight ascus is shown containing eight ripe (black) ascospores. The curved sac to the right also contains eight ripe ascospores. In contact with the tip of the curved ascus is a sac containing four unripe (slightly colored) spores; the other four nuclei have probably disintegrated. Next to this is an ascus in which the first four spores (from the tip) are gray (nearly ripe), the next two are colorless (unripe) and the last two are black (ripe). Each pair of spores (1 & 2, 3 & 4, 5 & 6, and 7 & 8) is genotypically identical, since the third nuclear division is equational. The failure of Spores Nos. 5 and 6 to ripen in the above ascus was determined by genetical factors. In genetical studies each ascospore is grown in a separate tube. The segregation of characters from a single zygote can be "read off" directly by inspection of the resulting (haploid) mycelia. Magnification about 200 diameters.

of allels is so great that they are segregated at random this limiting value is attained. When two loci are so close together that no double crossovers occur between them, the ratio expresses the true genetical distance between them in morgans. The same holds for calculating short distances between a locus and the s.f.a. by the ratio:

$$\frac{\text{second-division segregations}}{2\ (\text{first-division segregations plus second-division segregations})}$$

However, the limiting value of this ratio is .33.

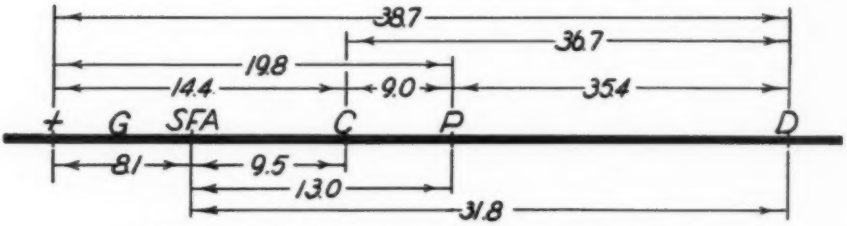
Spores are arranged in asci in six ways. When the distance of the locus from the s.f.a. is so great that a sufficient number of crossovers occurs to give a random result, there will be two asci with the arrangements that could be interpreted as no crossover between the locus and the s.f.a. (first-division segregation of the allels: *aaaaAAAA, AAAAaaaa*), to four asci that could be interpreted as a single crossover between the locus and the s.f.a. (second-division segregation: *aaAAAAaa, AAaaaaAA, AAaaAAaa, aaAAaaAA*).



SPORE ORIENTATION EXPLAINED AS THE RESULT OF CROSSOVERS

Figure 3

Table III shows the observed arrangement of spores in asci as determined by actual observation of separately planted ascospores. This drawing shows the way in which these results could be brought about through crossing over at the points indicated in the drawing. Thus at *a* the (+) sex factor is associated with *crisp* (C) and with the normal allelomorph of *pale* (p) in four of the spores (noncrossover association). The other four are −cP, also noncrossovers. In *b* one of the four pairs of spores is +CP and another pair is −cp due to a crossover between the Cp/cP loci. There are also two noncrossover pairs of ascospores (+Cp and −cP). The other diagrams show how the observed situation (Table III) may result from crossovers at the points indicated. From these observations it is possible to deduce the order and approximate position of the genes (See Figure 4).



MAP OF THE SEX CHROMOSOME OF NEUROSPORA

Figure 4

Genetical distances in the sex-chromosome determined by data submitted in this paper. All are direct measurements and none is the result of addition or subtraction of two other values. The gene *gap* (G) has been previously (Lindegren[9]) shown to be about 4 units to the left of the spindle-fiber attachment (SFA). Although there are discrepancies due to sampling errors concerning actual distances, all data agree on the seriation of the six loci: the sex-differentiating allels (+/−), the gene *gap* (G), the spindle-fiber attachment (SFA), and the genes *crisp* (C), *pale* (P), and *dirty* (D).

This results in 67 per cent second-division segregation or an apparent distance of 33 morgans from the s.f.a. for the locus in question. Most of the genes encountered in the analysis of *Neurospora* asci are so far from the s.f.a. that the ratio:

$$\frac{\text{second-division segregations}}{2\ (\text{first-division segregations plus second-division segregations})} = .33$$

This means that they are too far from the s.f.a. to work with unless other genes between them and the s.f.a. can be found. Stocks carrying these genes are put aside to be tested against stocks carrying genes close to the s.f.a. The gene, *dirty*, falls into this category. There are so many crossovers occurring between *pale* and *dirty* that no attempt will be made to analyze crossing-over in this region until another allel has been discovered between *pale* and *dirty*.

In analyzing the segregation of allels in *Bombardia lunata*, Zickler[11] discovered that the allels which he studied were segregated at the second division in 67 per cent of the asci. He interpreted this as indicating that the s.f.a.'s split at the first division and segregated at random. As evidence for random segregation of the s.f.a.'s, these data have only a negative value. It seems to the writer more probable that Zickler was dealing with allels more than 33 morgans from the s.f.a. and that the s.f.a.'s were segregated reductionally at the first division.

The distances in the map can be corrected by consideration of the cross-

TABLE III. Arrangement of Spores in Asci

						Strands	+ and s.f.a.	s.f.a. and C	C and P
a	−cP	−cP	+Cp	+Cp	45	180	0	0	0
b	−cP	−*cp*	+Cp	+*CP*	9	36	0	0	18
c	−cP	−*Cp*	+Cp	+*cP*	7	28	0	14	0
d	−cP	+cP	−Cp	+Cp	6	24	12	0	0
e	+cp	−cP	+Cp	−CP	2	8	4	0	4
f	+Cp	−cP	+Cp	−cP	1	4	2	2	0
g	+cp	+Cp	−CP	−cP	1	4	0	2	2
h	−Cp	−Cp	+cP	+cP	1	4	4	0	0
i	−cP	+Cp	−Cp	+cP	1	4	2	2	0
j	+CP	−CP	+cp	−cp	1	4	2	0	4
k	−cp	−cp	+CP	+CP	1	4	0	0	4
l	−cp	+Cp	+CP	−cP	1	4	2	2	2
m	−Cp	−CP	+cP	+cp	1	4	4	0	2
n	+CP	+cP	−cp	−Cp	1	4	0	2	2
					78	312	32	24	38
Morgans							10.2	7.7	12.1

overs which have occurred in the three regions lying between: (1) (+) and s.f.a., (2) s.f.a. and *C*, and (3) *C* and *P*. The arrangements of the spores in the asci are shown in Table III.

Since the last division is a simple mitosis, each pair of spores is represented by one symbol. The first column of figures shows the numbers of asci in the different classes. Multiplying this number by four gives the number of strands or chromatids involved. In the diagram (Figure 3) the various orientations are interpreted as the results of crossovers. In the last three columns the number of crossovers occurring in each region are shown and the distances recalculated by dividing the number of crossover chromatids by the total number of chromatids.

Two-strand and four-strand double exchanges seem to be more numerous than three-strand double exchanges. This was also the case in the series reported in a previous paper (1936) but the numbers are not yet large enough to enable one to draw definite conclusions concerning the relative frequencies of the different types and it is possible that crossing-over in heteromorphic chromosomes (see below) may differ from crossing over in chromosomes that are not heteromorphic.

Sexual Cycles

The continued use of the term "sex-chromosome" may seem to require explanation since it is opposed to the "sterility-factor" concept (Ames[1]). The writer[8] has already pointed out that the terms (+/—) have an advantage over the letters (*A*/*a*). Letters are used to indicate single genes, at least as closely as that is possible. A study of what appeared to be true sterility factors in *N. crassa*[8] showed a marked difference between this type of factor and the (+/—) pairs of "allels." Dodge[5] has also described true sterility factors in Neurospora. No mutations of sex have been recorded in a study of more than 20,000 single ascospore cultures. This is in marked contrast to the mutability of factors determining the production of clamp-connections in the Hymenomycetes. Matsuura and Gondô[10] have shown that there are a pair of heteromorphic sex-chromosomes in the Ascomycete, *Peziza subumbrina*. Genetical analysis of the sex-chromosome of *N. crassa* has shown that there are a number of loci to the right of (+), but none has been discovered left of (+). This indicates (and, if subsequent data follow this rule, will prove) that the (+/—) pair of "allels" are not really allels, but loci at which the two sex-chromosomes become heteromorphic, and to the left of which no crossing-over occurs. The heteromorphism might not be obvious from a study of metaphase plates, just as translocations in Drosophila are not always detectable cytologically by study of metaphase plates. Heteromorphism would prevent crossing-over in the heteromorphic regions either through a failure of synapsis of non-allelic loci or because at this point one of the pairs of chromosomes is abbreviated.

Stages in Sex-Differentiation

Hanna's[6] description of multiple sexes in Hymenomycetes first raised the question of the general applicability of anthropomorphic concepts of sex. The writer will attempt to define a sexual cycle and to describe different types of sexual cycles in fungi. The essentials of a sexual cycle are the production of two gametes which fuse to produce a zygote that undergoes reduction and produces gametes thus completing the cycle. The gametes may range from single chromosomes to highly specialized sex cells produced by antheridia and oogonia. In the latter case sex functions through the action of sex organs; in the former case sex may function without the production of sex organs. The gametes may be genetically identical or may be differentiated genetically as follows:

1. Simple allelic or other mutational differentiation concerning somatic characters.

2. Allelic or other mutational differ-

entiation affecting the production of viable progeny, sterility rather than sex factors.

3. Allelic factors (one or two pairs) differentiating the sexes. This is the type of sex factor found in the Hymenomycetes. Sex factors differ from sterility factors in being constantly present throughout the species and in being the primary factors concerned with the production of the zygote which is always heterozygous for them. When two pairs of allels differentiate the sexes, four sexes are produced. Because simple allels are responsible for differentiating the sexes, many mutations are possible and since the only requisite is that the zygote be heterozygous for the sex factors, a multiplicity of sexes results from mutations.

4. Heteromorphic chromosomes with relatively large regions and many genes concerned in the differentiation of the sexes, so-called sex-chromosomes. In this case the two sexes would be relatively stable. This is probably the type found in some of the heterothallic Ascomycetes. Differentiation of types (1) or (2) may both be superimposed on either types (3) or (4).

In some sexual cycles, the gametes are genetically identical and no sex organs are formed. The gametes may be nuclei containing one or more chromosomes or free cells containing one or more nuclei. In *Bacillus subtilis* (Badian[2]) gametes are single undifferentiated chromosomes produced in a single cell by the division of a single preexisting chromosome. These chromosomes fuse to form the zygote which immediately undergoes reduction to produce four single chromosomes. Three of these degenerate and the endospore is produced about the fourth. Thus a completely undifferentiated sexual cycle is possible without either sex organs or sex factors. In this simple sexual cycle, sex cannot function to *reassort* genes, but it may *rearrange* them by meiotic and chromosomal upsets. But the production of a heterozygous zygote might result if a cell fusion preceded the chromosomal fusion.

In the yeasts, the gametes are separate but differentiated cells (*Saccharomyces octosporus*) and the zygote results from the fusion of two of these cells. It has not been ascertained if these gametes are always differentiated from each other. But because the gametes are free cells, fusions between genetically different forms are possible and sex could serve to reassort as well as to rearrange genes.

In the Hymenomycetes there are no sex organs. Hyphae with nuclei differentiated by one or two pairs of allels (type 3) fuse and eventually produce fruiting bodies. The zygotes are heterozygous for the sex differentiating allels. In this sexual cycle the sexes are genetically differentiated, but otherwise cannot be distinguished.

In the Zygomycetes, the sex organs are merely enlarged ends of hyphae. In some species they are undifferentiated (isogamous). In others, male and female sex organs can be distinguished by difference in size. In many of the Zygomycetes the sexes are differentiated by stable sex factors. The stability of these sex differentiators suggests that the differentiation is by heteromorphic chromosomes (type 4) rather than by a single pair of allels. Upon this type of genetical differentiation, types (1) and (2) are probably frequently superimposed. The distinction between male and female sex organs is doubtless a direct expression of the action of the sex factors. Therefore, the gross difference between the sex organs is the reflection of the genetical difference between the nuclei in the different kinds of thalli (+ and —).

In the Phycomycetes well developed sex organs are produced but a genetical analysis is not available. In some of the Ascomycetes sex organs are as clearly differentiated as in the Phycomycetes. Genetical analysis has revealed two different situations. (1) In Pyronema, each ascopore, which contains a single haploid nucleus, gives rise to a thallus on which both types of sex organs are produced. Differentiation of the sex organs is not dependent upon

genetical factors, but is similar to the differentiation of sex organs occurring in hermaphroditic plants. In Pyronema sex organs result from gross structural differentiation and neither sex factors nor sex-chromosomes are features of the sexual cycle. (2) In *Pleurage anserina*, Ames[1] has described both male and female sex organs. The nuclei are of two genetically stable types (+/—). Spermatia from a (+) thallus will not fertilize oogonia on the same type thallus, (+), but will fertilize oogonia on a (—) thallus and *vice versa*. Dodge[4] has suggested that a similar condition exists in Neurospora. Although the cytological details are not complete in either case, the analysis seems fairly satisfactory and in line with what is known in other forms. As already pointed out, it seems probable that the genetical factors are not simple allels but sections of chromosomes containing many factors. In this case they cannot be considered sterility factors, but are true sex differentiators. Therefore, in these life cycles, sex is differentiated both by genetical factors (sex chromosomes) and by gross structural differentiation (antheridia and oogonia). Although the sex of an organ is obvious from its structure, (+) and (—) nuclei cannot be distinguished as to maleness and femaleness. This is analogous to the fact that male-producing sperm (or eggs) cannot be distinguished from female-producing sperm (or eggs) as to maleness and femaleness. In Drosophila males are of XY constitution, while in chicken the situation is reversed, the females being of XY constitution.

With rare exceptions principally in the Myxomycetes, the zygote nucleus is the only diploid nucleus in a fungal life cycle. Each mutation is easily expressable and advantageous mutations are rapidly selected. But complex structures are not possible because they require phenotypic uniformity. This stability is obtained in higher plants and animals by diploidy which opposes each gene with a normal allel and the dominance of the normal allel prevents change in the phenotype. In the Basidiomycetes uniformity of the phenotype is maintained by the dikaryotic nature of the tissue. Each cell contains two nuclei of opposite sex and the dikaryon is maintained by clamp connections. This type of tissue makes possible the complex fruiting bodies of the Hymenomycetes which are the most elaborate structures found in the fungi.

This analysis shows that neither genetical differentiation nor sex organs are essential to a sexual cycle. However, either or both may be characteristic features of a given cycle. Furthermore, either sex organs or genetical differentiation of the gametes may be the sole criteria by which the sexes are distinguished. In primitive sexual cycles there were probably neither sex organs nor sex chromosomes. Sex chromosomes probably developed as a result of meiotic accidents followed by transgenations. The absence of sex chromosomes in most Ascomycetes suggests that this simple type of sexual cycle has a high survival value.

Summary

1. Genetical data suggest that the sex chromosomes of *Neurospora crassa* are heteromorphic.

2. Classification of sexual cycles of fungi indicate that sex factors, sex chromosomes, and sex organs are not essential to a sexual cycle and are probably recent phylogenetical developments. Opposite sexes may exist and be distinguishable although they cannot be classified as to maleness and femaleness.

Literature Cited

1. Ames, L. M., 1934. Hermaphroditism involving self-sterility and cross-fertility in the Ascomycete *Pleurage anserina*. *Mycologia*. 26: 392-414.

2. Badian, Jan, 1933. Eine cytologische Untersuchung über das Chromatin und den Entwicklungszyklus der Bakterien. *Archiv f. Mikrobiologie*, 4: 409-418.

3. Dodge, B. O., 1930. Material for Demonstrating Sexuality in the Ascomycetes. *Torreya*, 30: 35-40.

4. ————, 1932. The Non-sexual

and the Sexual Functions of Microconidia of Neurospora. *Bull. Tor. Bot. Club,* 59: 347-360.

5. ————, 1935. A Recessive factor Lethal for ascospore formation in Neurospora. *Bul. Tor. Bot. Club,* 62: 117-128.

6. HANNA, W. F., 1925. The Problem of Sex in *Coprinus lagopus. Annals of Botany,* 39: 431-457.

7. LINDEGREN, CARL C. 1933. The genetics of Neurospora—III. Pure bred stocks and crossing-over in N. crassa. *Bul. Tor. Bot. Club,* 60: 133-154.

8. ————, 1934. The genetics of Neurospora V. Self-sterile bisexual heterokaryons. *Jour. Genetics,* 28: 425-435.

9. ————, 1936. A six-point Map of the Sex-Chromosome of *Neurospora crassa. Jour. Genetics,* 32: 243-256.

10. MATSUURA, HAJIME and ANBU GONDO, 1935. A Karyological Study on *Peziza subumbrina* Boud., with Special Reference to a Heteromorphic Pair of Chromosomes. *Jour. Fac. Sc.* Hokkaido Imperial University, 3: 205-217.

11. ZICKLER, H., 1934. Genetische Untersuchungen an einem hetero-thallischen Askomyzeten (*Bombardia lunata* nov. spec.) *Planta,* 22: 573-613.

✦

LINKAGE OF PINK-EYE AND ALBINISM IN THE DEER-MOUSE

FRANK H. CLARK

Laboratory of Vertebrate Genetics, University of Michigan

PRELIMINARY results show that the pink-eyed mutation, *pallid,* is linked with albinism in the deer-mouse, *Peromyscus maniculatus.* Sumner[4, 5, 6] reported the origin, appearance, and apparent linkage between these characters but he did not attempt to work out the crossover percentage. For reasons given later, I shall designate the pink-eyed mutation in the deer-mouse as *pink-eye* or *pink-eyed dilution* and discontinue the use of the name, *pallid.*

In the present study, pink-eyed and albino deer-mice, derived from Dr. Sumner's stock, were mated together. The F_1 wild-type offspring were crossed with double recessive pink-eye—albino mice. If no linkage occurs between these characters, we should expect four types of offspring to appear in equal numbers: viz., agouti, pink-eye, albino, and pink-eye—albino. No attempt was made to distinguish between albinos and pink-eye—albinos which are phenotypically the same. The backcross offspring thus far obtained are as follows.

(cross-over) agouti	(parental) pink-eye	(parental) albino	(cross-over) pink-eye—albino	Total
12	62	78		152

The percentage of crossing over obtained by dividing the normal agouti crossover class (12) by one-half the total number (76) is 15.8 ± 1.99. The deviation of this crossover percentage from that expected on a no-linkage basis is 34.2 ± 3.39 per cent. This deviation is over 10 times its probable error and, therefore, highly significant. The genes for pink-eye and albinism in the deer-mouse evidently lie then in the same chromosome approximately 16 crossover units apart. Except for two or three litters, the backcross young were obtained by mating F_1 doubly heterozygous females with double recessive males. No data are yet available to show whether or not crossing over is less frequent in the male deer-mouse than in the female.

Resemblance Three Rodent Genera

The crossover percentage between the genes for albinism and pink-eyed dilution in the house-mouse (*Mus musculus*) as calculated by Castle and Wachter[1] from their own data and those of Dunn[3] is 16.44 ± 0.82 in the female, and 13.77 ± 0.57 in the male. Linkage between pink-eye and albinism in the Norway rat was first reported by Castle and Wright[2] and studied further by Dunn.[3] The crossing over is about 21.9 per cent in females and 18.4 per

cent in males. A third gene in the rat, red-eyed yellow, lies between albinism and pink-eye on the same chromosome and is very closely linked with albinism, the crossover percentage being less than one. Sumner[7] (p. 33) believed that the *pallid* character (as he called it) in the deer-mouse was probably homologous to red-eyed yellow in the rat. I have shown, however, that the crossover percentage between this character and albinism is nearly the same as that existing between pink-eye and albinism in both the Norway rat and the house-mouse and not at all like that found between red-eye and albinism in the Norway rat. For this reason, I have discontinued the name *pallid* and substituted the name *pink-eye* even though many pink-eyed deer-mice are really more nearly red-eyed. Such variation in eye pigmentation might easily be due to modifying genes.

In the house-mouse, the chance that a gene will lie on any particular chromosome pair is one in 20, since there are 20 pairs of chromosomes in this rodent. The chances that two genes will lie in the same chromosome is obviously $1/20 \times 1/20$ or one chance in 400. The Norway rat and deer-mouse possess 21 and 24 pairs of chromosomes respectively, and the odds are slightly less than 1 in 400 that any two genes in those species will be on the same pair of chromosomes. Since pink-eyed dilution and albinism are linked and have nearly the same amounts of crossing over in the Norway rat, the house-mouse, and the deer-mouse, it seems highly probable that these characters are homologous in these three rodents and that one homologous pair of chromosomes may have been derived in these three genera from a common ancestor.

Literature Cited

1. Castle, W. E., and W. L. Wachter. 1924. Variations of linkage in rats and mice. *Genetics*, 9.

2. Castle, W. E., and S. Wright. 1915. Two color mutations of rats which show partial coupling. *Science*, 42.

3. Dunn, L. C. 1920. Linkage in mice and rats. *Genetics*, 5.

4. Sumner, F. B. 1917. Several color mutations in mice of the genus Peromyscus. *Genetics* 2:291-300.

5. ————. 1918. Continuous and discontinuous variations and their inheritance in Peromyscus. *Am. Nat.*, 52:177-208, 290-301, 439-454.

6. ————. 1922. Linkage in Peromyscus. *Am. Nat.*, 56:412-417.

7. ————. 1932. Genetic, distributional, and evolutionary studies of the subspecies of deer-mice (Peromyscus). *Biblio. Genetica*, 9:1-106.

✦

$1,000 FOR A POPULAR SCIENCE MANUSCRIPT

A CASH award of $1,000.00 is offered by The Williams & Wilkins Company for the best manuscript on a science subject, presented before July 1, 1937.

Literary prizes are relatively common, but it is not so usual for a publisher to be bidding for science material in this manner.

The publishers put no limitations on the subject-matter or manner of handling, and none on the eligibility for the award. The MS. must be in English and "of a sort calculated to appeal to the taste of the public at large." The desired length is given as 100,000 words.

While any MS. on a science subject will be considered, it is expected that the author will prove to be a man or woman engaged in a scientific pursuit and who is possessed of the requisite literary skill to interpret science for that portion of the public which reads books.

To assure authenticity, the publishers have enlisted the services of some 25 or 30 "advisers," these being men of science of wide reputation and assured competence. One or more of the advisers will pass upon each MS. from the viewpoint of soundness and accuracy.

The award will lie in the joint discretion of four judges selected with a view to their especial qualification in choosing the sort of book that will appeal. These are:

Dr. Joseph Wheeler, Librarian of the Pratt Library in Baltimore, and chairman of the Book List Committee of the Association for the Advancement of Science; Harry Hansen, reviewer and critic for the New York *World Telegram* and *Harpers Magazine;* Dr. Lyman Bryson, Professor of Education of Teachers College, Columbia, and Director of the "Readability Laboratory"; and David Dietz, science editor of the Scripps-Howard newspapers.

Further details concerning the award may be had by addressing the publishers at Mount Royal and Guilford Avenues, Baltimore, Md.

A CHALLENGE TO THE EUGENIST

J. P. Scott
Wabash College, Crawfordsville, Indiana

In a recent article in the Journal of Heredity Cook[1] has criticized the current program of the American Eugenics Society, chiefly on the ground that what the eugenics movement needs is not mere facts but motivation, not knowledge but Messiahship. There is much justification for this point of view. Most advocates of racial improvement are far from fanatical, and there has been to date no case of an eugenist offering himself up to be sterilized as a public example.

On the other hand, it has recently come to the notice of the author that a more or less garbled version of the eugenic program is being spread throughout the educated section of the general population. It now seems to be a general impression among the college bred that eugenics, including sterilization, is a good thing for people who do not go to college. With the assistance of able propagandists (not lacking today) there is thus a reasonably imminent possibility that a Messiah of Eugenics might get something started. But the program which he should follow is far from being obvious.

A fair and general statement of the eugenic premises and program, acceptable to the majority of those interested, might run as follows:

1. Differential fertility between occupational classes exists.

2. General social fitness is to a high degree correlated with occupational class as arranged on a scale of amount of wages received.

3. General social fitness to a large extent varies with heredity.

4. The trait of general social fitness is therefore being gradually eliminated from civilized races by a process of artificial selection.

5. The advancement of civilization is desirable.

6. The advancement of civilization is dependent upon the advancement of social fitness, or at least upon maintaining it at a certain level.

7. It therefore becomes necessary to start a program of counter artificial selection, both against the socially unfit (negative eugenics) and in favor of the socially fit (positive eugenics).

Statements 1 and 5 can be accepted almost without question. To statement 3 must be added that general social fitness also varies with environment, to an extent not determined. It will be noted that these are statements of fact and opinion which by themselves do not point to a course of action.

The second statement is entirely open to question. In the first place there is no general agreement as to what social fitness is. Geniuses, who certainly contribute largely to civilization, often seem to owe their success to fortunate combinations of ability and disability. If fitness is defined in the simple terms of Natural Selection it becomes the ability to survive under a variety of conditions. Selection on such a basis would tend to eliminate the extremes of species, the saviours and imbeciles together. *Social* fitness must be defined with regard to the condition of the species at a particular time, and as promoting in general the welfare of the species at that time. It is therefore a variable in time and space.

Accepting this definition it does not automatically follow that general social fitness is highly correlated with economic status, and indeed it provides a problem of description difficult of solution even by scientific means. The second proposition above assumes that the ability to achieve power is identical with social fitness, such ability being highly correlated with financial success. It is not necessary to admit the primary

assumption. Are there no poor unworldly Rockefellers, no meek but honest Mussolinis? Is the "working class" barren of every talent as it apparently is of financial ability? If so, it very properly should be sterilized, and let us be done with social insurance.

The Alleged Elimination of Ability

It is true that there appears to be a considerable correlation between the abilities of parent and offspring, and there is every reason for assuming that part of it is caused by common heredity. It has been the basic contention of the eugenist that such primarily mental ability, forming a vital portion of general social fitness, is eliminated from civilized races by a process of selection akin to that employed by stock breeders. Other portions of general social fitness, such as fecundity and physical vigor, do not seem to be lacking in the "lower" class. The poor, as far as one can judge from history, have always outbred the rich in spite of an unfavorable environment. Even at our present peak of civilization we cannot claim that we are pampering the poor in lovely slums.

The crucial point in the eugenist's theory is therefore the disappearance of mental ability under civilization. There is not sufficient data at hand to establish this point. Historical assertions can too easily be accounted for by the common human tendency to revert to childhood in times of stress. A dead ancestor in common talk is always better than a living descendant. Even Galton, who first became alarmed over the extinction of the British peerage, was not able to show that England had suffered much from the lack of descendants of courtiers and beer barons. All modern scientific data are open to the objection of uncontrolled factors. The increase in number of injurious traits must be weighed against improvement in the technique of discovering those traits. An increase in the general rate of insanity may be accounted for as being the result of the increased life span, as may certain other diseases.

That the advancement of civilization is dependent upon advancement of social fitness is obviously untrue if the result of civilization is a decline in social fitness. Civilizations *have* advanced, and over long periods of time. That a decline in civilization is accompanied by a decline in general social fitness in the population is a proposition which can only be approached historically, and history is notoriously open to a variety of interpretations. Anatomical evidence indicates that man has changed very little if any in the last five thousand years. Was the brief spark of Italian Renaissance civilization extinguished by a sudden decline in the supply of native ability, or by the discovery of an ocean route to Asia? Civilizations are obviously correlated with material prosperity. It can well be argued that particular types of civilizations are dependent upon particular environmental conditions, which may not be permanent—that the sack of Rome was the inevitable end of a people who subdued their neighbors by force. Or that mechanized hari-kari is implicit in a civilization supported but not guided by science.

Finally, the practical program of the eugenics movement is open to question. Eugenists, accepting Natural Selection, at first assumed that the unfit could easily be eliminated by sterilization but were later confounded by *Drosophila* genetics, which indicated that the majority of injurious characters (in the fruit fly) were controlled by recessive factors. It is a hard thing to sterilize a heterozygote. But, as Muller[3] pointed out, and which is evident in any catalog of human heredity, the majority of human diseases reported to show Mendelian inheritance are dominant over the normal. Admitting the inconclusive nature of most data on human heredity, the whole feasibility of negative eugenics rests upon this question of fact.

Positive Eugenics

Positive eugenics is similarly bound up with the facts of evolution. Dar-

win, arguing by analogy from plant and animal breeding, accounted for evolution by Natural Selection. Fisher[2] assuming natural selection to be the sole cause of evolution, has postulated that continued selection must therefore lead to continued improvement in a breed or race. This does not agree with the facts put forward by taxonomists, who find that the differences between closely related species are in general non-adaptive and hence not subject to natural selection. Later Wright[4] pointed out that inbreeding, combined with certain other factors, will lead to the type of evolution found in nature. He further concluded that selection for general adaptive value will lead eventually to a full stop in evolution with the acquisition of homozygosity. That is, selection can produce only relatively minor and impermanent changes in a race, an idea borne out by the rapid reversion of domestic breeds of animals placed under wild conditions.

On the difference between the Fisher and Wright theories of evolution rests the practicability of positive eugenics. If the eugenist is to improve the race by selection it seems likely that he will have to borrow the technique of animal breeding: to cross-breed and then inbreed with selection for *particular* characters. This would lead to a sort of caste system in human races, which according to entomologists has some favorable aspects.

In fairness to the eugenist it should be pointed out that even if all the above objections should be valid he still has served a useful function. The idea of selecting a superior mate usually results in marrying one with similar tastes and interests, leading to personal happiness if not racial progress. With family control the stupid person will ruin his children by education if not by inheritance. Further, in a modern, crowded and long-lived generation fecundity becomes a less important part of social fitness, and it is perhaps just to ask the poor to have less children and more fun—*given the opportunity*. The obvious reason for the differential birth rate is that the discomforts of pregnancy are minor troubles to the poverty-stricken. Perhaps the soundest way to correct the differential birth rate would be to level out economic conditions.

The greatest weakness as well as the strongest motivation of the eugenist has been his wish to improve permanently the human race rather than to be satisfied with temporary alleviation of environmental conditions. It is based on the Weissmanian concept of the imperishable germ plasm, which does not take into account the impermanence of particular and personal gene complexes. A parent can hand on only half his heredity to a single child, and a parent of four will on the average fail to pass along one-sixteenth of the genes possessed in a heterozygous condition. That is, the mechanism of biparental inheritance makes impossible the preservation of the personality of the most valued genius, and even of his entire heredity in mixed form without an infinite number of children. Particular gene complexes can only be preserved by strict inbreeding of homozygous individuals, and there is no indication that the most desirable human types come from such "pure lines."

If it is not desirable to divide the human race into a number of inbred castes, then the relationship of eugenics to heredity becomes a minor one, but the movement might well be carried on to the study of civilization in general. The eugenist would then function as a student and seer of civilization, not so much a Messiah of the germ plasm as an enlightened racial weather prophet, attempting to ease the shock of transition from one form of civilization to the next. But before trusting him to guide us either up the evolutionary tree or into the sociological soup, the author, as a sceptic and scientist, would require the following *facts* from the research division.

1. Is general social fitness highly correlated with economic condition?
2. Has the differential birth rate actually produced deterioration of the

human race in general social fitness?

3. If there is such an effect, has it had an adverse effect upon civilization?
4. Is inbreeding or natural selection the more important factor in evolution?
5. Are the majority of injurious human traits controlled by dominant, recessive, or multiple factors?
6. Can the level of *general* social fitness be raised by a stock breeding program?

Literature Cited

1. Cook, R. 1936. A Eugenics Program. *Jour. Hered.* 27: 195-201.
2. Fisher, R. A. 1930. The Genetical Theory of Natural Selection. Clarendon Press, Oxford.
3. Muller, H. J. 1935. Human Genetics in Russia. *Jour. Hered.* 26: 193-196.
4. Wright, S. 1935. Evolution in Populations in Approximate Equilibrium. *Jour. Genetics* 30: 257-266.

FULL discussion of Professor Scott's "six points" would far exceed available space, so that brief comment must suffice. It may be admitted that present birth trends, though apparently culturally unfortunate because of the necessity of starting "back to scratch" in a large fraction of each generation, would be genetically desirable if there were a reasonably high *negative correlation* between social status and genetic constitution. If the above proposition is not true, present birth trends are not only culturally to be deplored, and may portend biological disaster—even if certain possible alternatives suggested by Dr. Scott were effectively applied. A hasty rumaging around in the writer's intellectual attic does not bring to light any recollection of instances in which a persistent negative correlation exists between phenotype and genotype, though the complications of dominance and environmental effects often reduce the positive correlation to considerably less than 1. He resists a temptation to enlarge on the perhaps hazardous speculation as to what would happen to a livestock breeder who decided to abandon selective efforts, basing his action on the fact that little exact genetic knowledge exists regarding the inheritance of economically desirable qualities in domestic animals.

The whole matter is so freighted with difficulties economic, sociologic, and biologic, that it begins to look as though the fungi, whose genetic pecularities Dr. Lindegren so ably discusses in our leading article, have the best of the bargain. Since it confines its diploid existence to a single cell in the life cycle, it is possible to tell merely by looking at our friend Neurospora what tale the fungi genes have to tell. In view of the battery of above mentioned complexities, perhaps the easiest solution is to persuade our friend Homo to consider the moulds and the mushrooms and to "go haploid." Just how we should go about this remains a mystery. Perhaps our writers could be "sold" on the possibilities of the multiplicity of sexes of which Dr. Lindegren speaks. This should furnish a welcome polygonal respite from the theme of the triangle, which is limited to three sides, and the sum of whose angles always equals two right angles. The resulting urge engendered in the hearts and minds of the reading public to haploidy and polysexuality might, by an obscure Lamarckianism result in a consumption so earnestly desired.

To regress from such airy speculation, there would seem to be little to be gained by fostering trends that obviously are working against all our efforts at social improvement. Perhaps the present writer is biased, but to him it seems that this situation is so omniously freighted with disaster that the eugenist, the educator, the physician, and the social worker could very well join forces to determine what changes are feasible. Accordingly, he suggests the problem of the development of socially effective eugenic measures as an excellent project for Dr. Scott's new Research Division. It would be a pleasure to furnish the palm-leaf bouquets to the person or the group that produces a workable plan for utilizing the agencies under social control to improve the racial qualities of *Homo sapiens Americanus*, (leaving the larger problem of the social improvement of *Homo sapiens Wideworldensis* for a future and a wiser generation). The persons who do this are certain to need plenty of inspiration and all the knowledge that can be had. No small-time propagandist will be able to do the job, although some of them have, and some of them will continue, to do their bit to befuddle very important issues. Perhaps other readers of the Journal already have a few well-seasoned planks in their intellectual attics. If such is the case it would be a privilege indeed to have the honor of displaying them to a waiting world.

Dr. Scott has himself suggested one plank in such a program, which plank seems akin to that presented with consideraable gusto in another communication published in this issue of the Journal, by Dr. H. J. Muller. The Editor has grave doubts whether this single plank contains enough lumber to build a raft to float an allegedly sinking civilization, but welcomes discussion of the subject. If a differential birth rate negatively correlated with productive intelligence is an essential feature of a competitive economic order, the proposed Messiah may have an even more difficult and dangerous task than we have supposed.—Editor.

A PAIR OF TUBERCULAR TWINS*

D. R. Atzenhoefer and D. Cecil Rife†
Department of Zoology, Ohio State University

ALIKE EVEN TO HAVING SIMILAR TUBERCULOSIS INFECTIONS

Figure 5

Twin *A* suffered an attack of tuberculosis a month later than Twin *B*, but has a slightly more severe involvement (see X-ray photographs). No cases of tuberculosis are known among the relatives. The probabilities, on the basis of other similarities, that these twins are identical are in the nature of 150,000 to 1.

ON January 6, 1936, a pair of twin boys was admitted to the Franklin County Sanitorium, Columbus, Ohio, for medical treatment. According to Rife's diagnostic formula‡ the odds are 153,599 to 1 in favor of their being monozygotic.

Both have active ulcero-caseous tuberculosis in the left lung, chiefly in the upper lobe, the condition of twin *A* being more advanced than that of twin *B*.

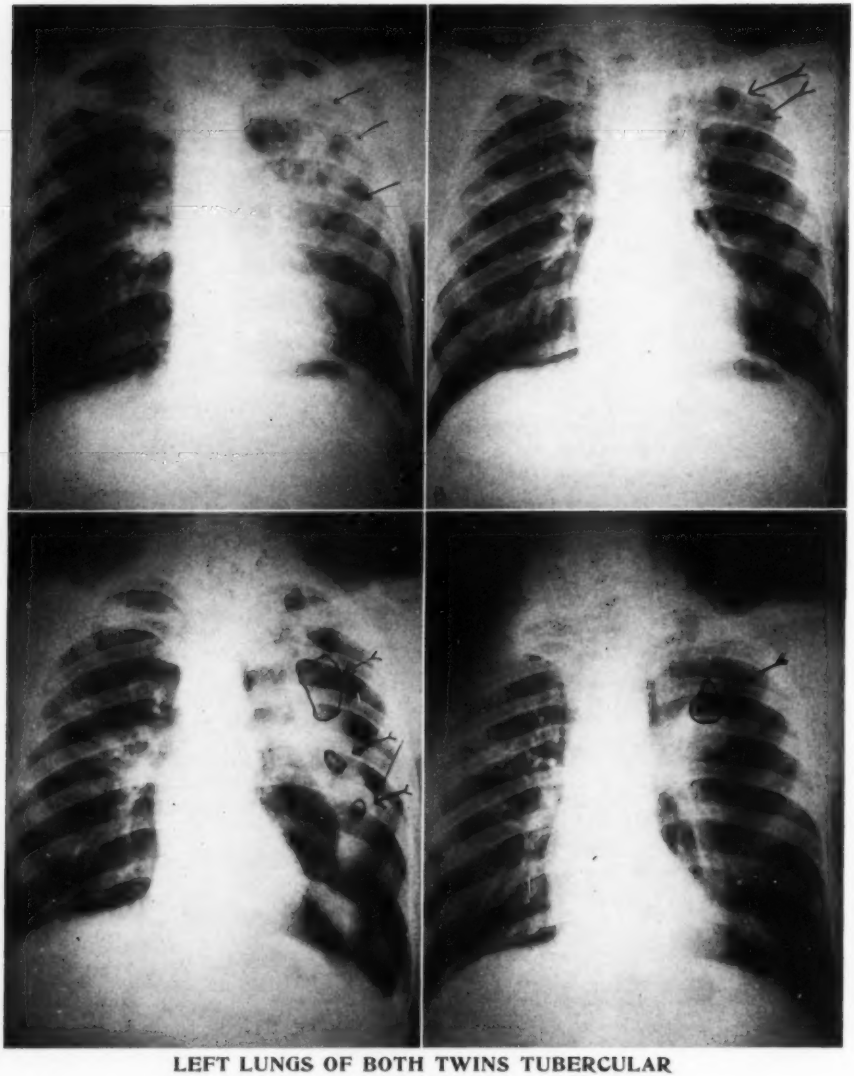
Twin *B* had an attack of pleurisy in December, 1934. In April, 1935, the family physician advised him of a tubercular "spot" on his left lung, and since that time he has performed no hard work.

Twin *A* became ill in May, 1935, and

*Genetic Studies of Monozygotic Twins, No. IV.

†The authors wish to express their appreciation for the splendid cooperation given by the following officials of the Franklin County Sanitorium in this research: Myron D. Miller, M.D., Elmer Kilpatrick, M.D., Emerson Blake, M.D., and Edith Atzenhoefer, R.N.

‡Rife, D. Cecil. *Journal of Heredity*, 24: 339. 1933.



LEFT LUNGS OF BOTH TWINS TUBERCULAR

Figure 6

Above are shown X-ray photographs of the twins just before pneumorthorax treatment. The arrows indicate the infected areas. Below are shown the condition after air had been admitted into the chest cavity (pneumorthorax). The infected areas are enclosed in heavy lines, and indicated by arrows. The location of the tubercular areas is remarkably similar in the two twins, carrying the resemblance of these almost certainly genetically identical individuals to one more fine detail.

began to lose weight; at that time he went to bed, gradually regaining strength and finally resuming regular activities. Again in August, 1935, he had an attack of pleurisy, at which time the family physician advised bed rest.

The accompanying X-ray photographs show by arrows the areas chiefly affected.

It is interesting to note that twin *A* shows a more extensive area of infection than twin *B*, yet became ill at a later date than did his brother. As shown by the photographs, the infected regions are very similar in location.

With the exception of about a year and a half, they have always lived together. No other cases of tuberculosis are known among their relatives.

Both boys are musically inclined and participate in the local popular orchestras.

✦

GENETICS AND POLITICS

To the Editor:

C. C. Little's article, *Bolshevik Genetics*, which I have only recently had occasion to see, raises a number of objections to the book by Mark Graubard, *Genetics and the Social Order*. The main complaint is that the book is written from the "partisan" standpoint of one who favors socialism, and that it is accordingly onesided, particularly in its attack upon the eugenists. And the unsuspecting reader, it is claimed, is not even warned of this partisanship at the beginning of the book.

The questions at issue are so important for geneticists as to call for some further comment here.

As to the matter of a prior warning, perhaps the reviewer did not read Graubard's introductory chapter, which states quite directly that "the state is in the hands of an exploiting class," and that as a result "bourgeois thinkers cannot face reality with a scientifically honest and free approach," while on the other hand it is stated of Marxism that "the very backbone of it is the scientific method." This would seem not only to make Graubard's position quite clear at the start, but also to show why this position should be considered as "partisan" by Little.

Biologists consider that they have the right to present the theory of evolution to their students as scientific fact, despite the objections of the Fundamentalists, who complain that the question is "controversial" and the biologists "partisan." In fact, the biologists are proud of such a "partisanship." Now, scientific socialists consider that they have a similar right to the direct statement of the socialist position as that representing scientific truth, despite condemnation or repression by the much more powerful group having interests vested in the present order. Those whose minds are not yet made up may sometimes find this situation aggravating, but in practical fact they probably will not be able to avoid a decision for long. Hence quite direct statements are needed.

Geneticists cannot stay aloof from the momentous questions here involved by seeking refuge in an ivory tower or in some *m*-chromosome, because genetics is today being dragged into the center of the conflict by the Fascists in their attempt to fabricate a theory to counter the socialist theory and to serve as a decoy to all who can be caught by it. If one will read for example some of the presidential addresses of the "Eugenics Research Association" of the U. S. A., or glance at the "Rassenpflege" propaganda of Germany, he will see that the "Eugenics" movements in these countries have become just what Graubard claims, although they profess to be derived from the science of genetics and thus involve the geneticists as their accomplices until the latter deny it. Graubard has performed a much needed service here in showing the reader that genetics can be disentangled from this disastrous perversion of it that is playing such a prominent part on the stage of present day events.

Those who have read in their context the sentences on topics of pure genetics which Little quotes from Graubard's book, will find them quite in harmony with modern genetic theory. But in the stress of these days of rapid social change something more than pure genetics alone is needed for the general reader. It is necessary for the relation to be shown between genetic facts and social facts, and this can be done only by one trained both in modern genetics and in modern social science, the latter, on our view, meaning primarily Marxism. Achieving such a synthesis he will show, as Graubard has, that only on the basis of a socially directed economic system can there be an adequate use made of the principles of genetics in the service of man. For only then will society be concerned in giving to each individual the fullest opportunity for development into a social being, in accordance with his genetic ability and needs. Only then, in fact, can genetic differences, now so overlaid by differences of social origin, be adequately recognized and taken account of. And only

then can we be properly guarded against the misuse, for class interests, of such tools as sterilization. Further, whatever can be done on the positive side of genetic improvement—and as the reader may know, the present author thinks this is a great deal—it certainly can be done properly only under socialized control. The matters here involved, which lie largely outside the scope of Graubard's book, will, as he says, be worked out scientifically and dealt with in the interests of society after these interests become the ruling ones. The only way to make possible a scientific approach to these matters is by first recognizing the truth of the primary contentions which Graubard puts forward. These constitute the burning immediate issues that have forced geneticists into such a pivotal position in the life of today. Read the literature of the Germany of today and you will see that hereon have been made to depend, in theory, at least, the questions of war or peace, of the future of civilization, in short, of whether evolution shall go backward or forward.

H. J. MULLER

The Editor welcomes the fullest discussion of the political implications of genetics. This does not imply, however, that he or the American Genetic Association accepts or endorses the Muller-Graubard view of the situation. If it is stupid to admit that the capitalistic system is very far from perfect (something that no intelligent person can deny) and yet to prefer to work toward a better order under that system, the Editor is certain that many readers of the JOURNAL are suffering from the same form of stupidity that he is afflicted with. Perhaps to a convert to Marxism, Graubard's book seemed objective and purely factual. To one who is not convinced that the patron saint of Bolshevism preached an inspired evangel, Graubard's book may seem as unscientific and as emotionally biased in one direction as are the writings and many of the actions of the Fascist group in another direction. To such stupid people the conflict has certain striking parallels to other religious conflicts (by whatever named called) that have dogged mankind through the ages. In these circumstances the social duty of all geneticists to "go Marxist" is not clear, though there is no doubt of the necessity and the social duty to demand, and to work under any system for the intelligent application of science to a multitude of social and political problems. There seem to be compelling reasons for doubting that this will be easy under any system.

Dr. Muller performs a very important service in calling to the attention of geneticists the disconcerting fact that they seem to have already been drafted willy nilly as major prophets of the two opposing philosophies of the right and the left. Their "pivotal position in the life of today" offers alarming possibilities of becoming such a hot spot that both *m*-chromosomes and ivory towers may require a deal of air-conditioning to keep them habitable.—EDITOR.

MARRIAGE IN THE COLLEGE CURRICULUM

225 Colleges out of 403 Deal with Some Aspect of the Subject—90 of 225 Teach Eugenics

A RECENT study of *Education for Marriage among American Colleges* by Cecil E. Haworth will presently be published as a dissertation. Some of the high points of Mr. Haworth's study were published in the *Bulletin* of the Association of American Colleges, November, 1935, pages 478-481. He examined the catalogs of 403 of the 465 member institutions in the Association of American Colleges and found in 225 colleges 234 courses which, in whole or in part, dealt with preparation for marriage and family life. By departments, the courses were offered as follows: sociology, 188; home economics, 38; religion, 4; psychology, 1; zoology, 1; general, 2. Later, Mr. Haworth sent questionnaires to the instructors in the 234 courses and 105 answered. From these he found many interesting facts, such as that 67 of the instructors are married, 45 are parents, 78 are men. In the 105 courses reported, the number of those with at least one class hour devoted to a special topic was as follows: divorce, 97; personal adjustments in marriage, 96; history of marriage, 96; eugenics, 90; legal aspects, 88; choosing mates, 86; philosophy of birth control, 85; comradeship, 84; careers for married women, 82; courtship, 74; child care, 68; recreation in the home, 68; religion in the home, 60. From ten to thirty per cent of the courses devoted one or more hours to such topics as engagements, weddings, honeymoons, petting, pregnancy, deviation of sex life, sex physiology, and other problems drawn from the general field of social hygiene. It is rather surprising to find 15 of 105 courses giving an hour to "technique of birth control," a topic which is illegal in many states and generally regarded as suitable only for medical schools. Moreover, there is general opinion of experts in social hygiene that reading, rather than class lectures, is a better method for handling this and similar social hygiene topics.—*Journal of Social Hygiene*, 22:29. January, 1936.

THE INHERITANCE OF SCREW TAIL IN CATTLE

"Kinky-Tail" Defect of Swine Paralleled in a Recessive Variation found in Inbred Red Polled Cattle

BRADFORD KNAPP, JR., M. W. EMMEL, and W. F. WARD*

THE inheritance of kinky tail in swine was recorded by Nordby[2] in 1934. A similar undesirable character, which has received the name of screw tail, has been found in the purebred herd of Red Polled cattle at the Government experiment station, Chinsegut Hill Sanctuary, Brooksville, Florida. This character appears similar to the character described in swine by Nordby. The calves are born with one or more distinct kinks in the lower portion of the tail. Such tails have the appearance of having been broken in one or more places. This character differs considerably from the wry tail character reported in Jerseys by Atkeson and Warren[1] in 1935. The wry tail character seems to affect only that portion of the tail near the root while the screw tail character occurs lower in the tail.

The Anatomy of the Defect

Screw tail is characterized by a fusion of one or more pairs of coccygeal vertebrae, the fusion being usually located near the end of the tail. The fusion may not be complete at the time of birth, but is completed during the first four to six weeks of life. This was true of Animal 1 as shown in Figure 8. The X-ray photograph (Figure 7*C*) of the tail taken at about three weeks of age shows that the vertebrae were not completely fused in the upper pair affected, but this same animal, when operated upon a month later showed completely fused vertebrae at this point (Figure 7*B*). No case has been found where more than two pairs of adjoining vertebrae were affected. Nordby reported that in swine three pairs of vertebrae have been found fused. In the calves born at the Chinsegut Hill station, three calves show the double kink while only one shows a single kink. Of the animals born in the North Carolina herd from which these cattle originated, one of the animals shows a double kink and three a single kink. The double-kinked tails are quite noticeably crooked.

Inheritance of the Character

The herd of Red Polled cattle at Chinsegut Hill Sanctuary comes from a highly inbred herd in North Carolina. In Figure 8 is shown a typical pedigree of one of four screw-tailed calves born at the station. This calf is sired by a screw-tailed bull and out of a cow some of whose offspring had shown the character.

The chart shows all of the animals in the pedigree that are known either to display the character or to carry it. There is, however, definite proof in some of the pedigrees of other affected animals that this character traces back to Sire 21 shown at the top of the chart.

Bull 2 shown in the chart has sired six calves from cows that are known carriers of the character. Of the six calves, three have been screw-tailed and three straight-tailed. This is the expected ratio from the back cross on a one-factor recessive hypothesis.

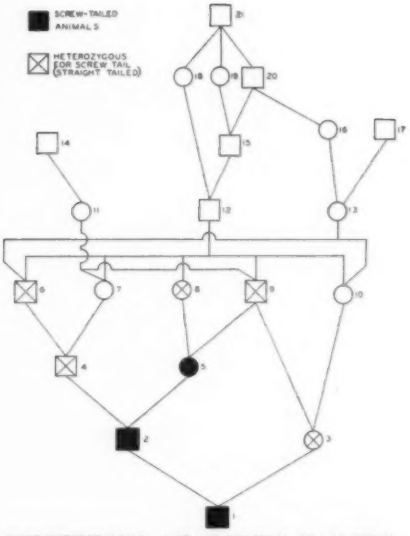
Further evidence that the character is probably recessive is shown by the fact that when Bull 2 was mated to other Red Polled cows that had not produced the character, he failed to produce any calves displaying the screw tail; and when mated with Florida Native cows in

* Bradford Knapp, Jr., and W. F. Ward are members of the staffs of the Florida Agricultural Experiment Station and U. S. Bureau of Animal Industry. M. W. Emmel is a member of the Florida Experiment Station staff.

INHERITED KINKY TAIL IN CATTLE

Figure 7

A shows the external appearance of the "screw tail" variation in cattle. This distorted effect is produced by two fused vertebrae (see X-ray photograph *C*). At *B* are two fused vertebrae taken from the tail of one of the calves born at the Brooksville station. This shows how the vertebrae are fused together to produce a kinky tail. This is very similar to the type of vertebral fusion found as an inherited characteristic in pigs and mice. *C* shows an X-ray photograph of the tail shown externally at *A*. The upper pair of vertebrae are not completely fused, while the lower pair are solidly grown together. Later when this animal was operated on the upper pair had become fused in the interval.



INBREEDING OF "SCREW TAILED" ANIMALS

Figure 8

Typical pedigree chart of one of the screw-tailed calves showing a considerable degree of inbreeding with relationship to a North Carolina herd to which all the screw-tailed animals studied in this report are related. The inheritance is evidently recessive, with other factors possibly modifying the number of vertebrae affected.

which the character had never been seen, the progeny were all straight-tailed.

The number of animals involved in this study does not completely explain the inheritance of the character. That it is recessive seems certain, since the screw-tailed bull when mated to straight-tailed cows of other breeding did not produce any animals that showed the defect. It would seem that a one-factor hypothe-

sis would best fit the inheritance of the character although there may be other factors involved that regulate the number of vertebrae affected.

The screw-tailed bull has also sired some cryptorchids, but there seems to be no linkage relationship between the two characters.

Summary

A screw tail or kinky tail character which has been found in Red Polled cattle, is ascribed to the fusion of one or more pairs of adjacent coccygeal vertebrae. The appearance is similar to that of kinky tail in swine.

The defect apparently is due to a single Mendelian recessive character, though other factors may be involved that regulate the number of vertebrae affected.

Literature Cited

1. Atkeson, F. W., and Warren, T. R. Inheritance of Wry Tail in Jersey Cattle. *Jour. of Hered.* 26:331-334. 1935.

2. Nordby, J. E. Kinky Tail in Swine. *Jour. of Hered.* 25:171-174. 1934.

✦

More About the Double Nasturtium Patent

To the Editor:

Whenever the term "basic" patent has been used in connection with Tropaeolum majus Burpeeii (Plant Patent No. 141), it has been in the sense of indicating the broader nature of the patent as compared with the ordinary plant patent. (See Journal of Heredity for Feb., 1936, and for May, 1936.)

It is true that the form of claim required by the Patent Office in plant patents is similar to that of design patents. The entire description and illustration is for all practical purposes the "claim."

What we have done in the present case is to cover in a single patent a range of colors rather than a single color. The different colors can be fore-determined and produced with precision when either of the two colors shown in the patent is available. No additional invention is involved in producing this additional range of colors. Anyone "skilled in the art" could produce these colors precisely and without inventive effort, if he possessed either of the two colors shown and described. Therefore, he would have no right either to claim these additional colors as inventions or to deprive the rightful inventor of these "super double" nasturtiums of the full "use" of his invention in its different color forms.

When a patent is granted on a design to be applied to a fabric, it is well understood that although the original design may be submitted to the Patent Office in black ink, the patentee is fully protected as to its use in red ink or dye or in any other color. A possible exception might be some new color which is itself covered by patent rights. In the case of Burpee's Super Double Nasturtiums, we limit ourselves to the range of colors indicated.

We do not designate by the term "variety" each color variation in which this "super double" nasturtium can be produced. To do so would require the recognition of hundreds of variations, one varying but slightly from the nearest neighbor in the color range. To cover each with a separate patent would be impracticable and to require this would be to defeat the very purpose of the plant patent amendment. Each of these separate colors is designated merely as a variant or sub-variety of the distinctly *new* variety on which U. S. Plant Patent No. 141 is granted.

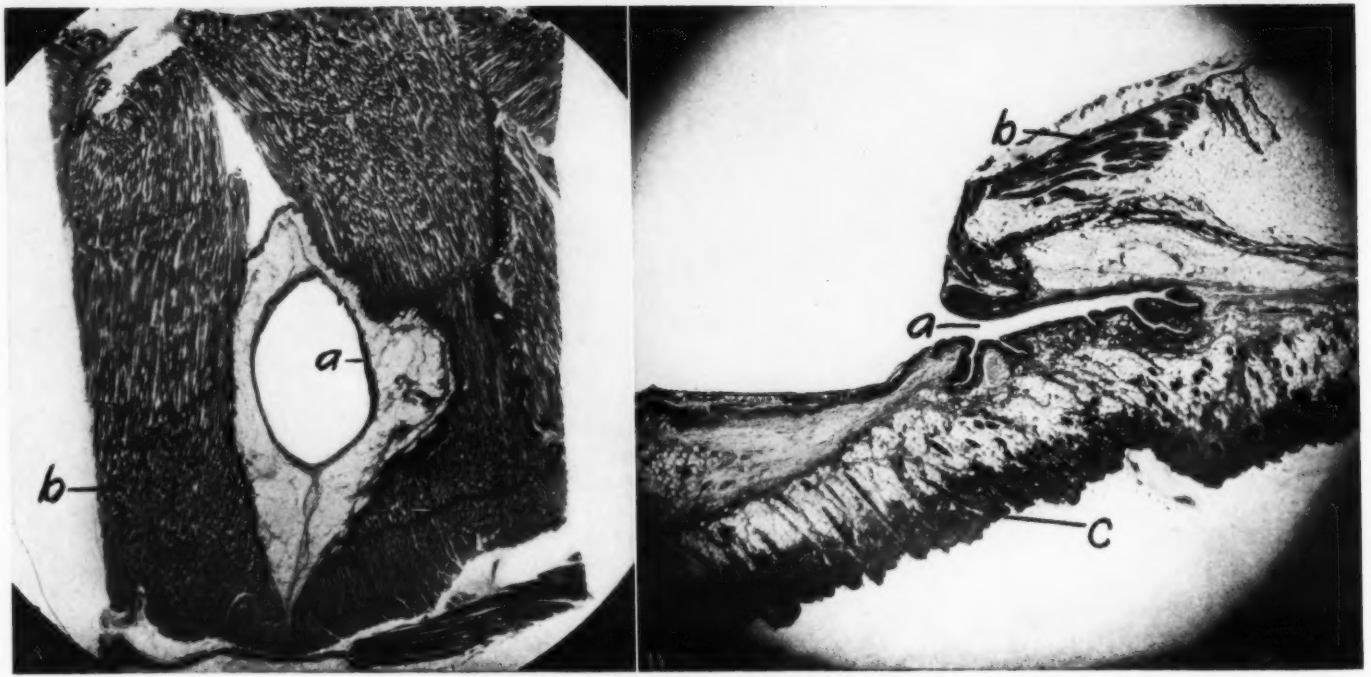
David Burpee

April 20, 1936

Which brings us right back to the questions: (1) Is a patentable variety the asexual progeny of a single plant or is it something else? (2) Does the present patent law include "genetic types"? (3) While plant patents follow design patents in having only one claim, it should be remembered that this claim *attempts* to follow the mechanical patent form, and that the plant patent amendment is a part of the mechanical-chemical patent law, not of the separate design patent law. While the logic of Mr. Burpee's view is admitted, there still remains the important question whether, as the law reads, Plant Patent 141 can be anything but an "ordinary plant patent." "The glorious uncertainty of the Law" holds the answer, and no other answer is final.

The question of the effect that these diverse views would have in "advancing science and the useful arts," which the Constitution sets forth as the purpose of the Patent Law, has interesting possibilities of speculation pro and con.

Too late to include in this issue we have received the text of a decision by the U. S. District Court for the Northern District of Ohio invalidating Plant Patent 110. Portions of this decision have a definite bearing on the questions discussed above. It is planned to discuss it in an early issue of the Journal.—Editor.



SECTIONS OF UMBILICAL HERNIAS IN RATS

Figure 9

A is a horizontal section of an umbilical hernia (6×). *B* is a photomicrograph (24×) showing a section through the axis of the hernial sac. The hernia appears as a ring *a* in the cross section (*A*). It appears as a sac (*a*) in the sagittal section (*B*). In both these illustrations *b* shows the abdominal muscle. The skin on the abdomen is shown at *c* in the right-hand photograph.

INHERITANCE OF UMBILICAL HERNIA IN RATS*

L. A. Moore and P. J. Schaible

Section of Dairy Husbandry and Section of Experiment Station Chemistry, Michigan Agricultural Experiment Station, East Lansing

UMBILICAL hernia occurs rather commonly in most species of mammals as well as in man. For example, in the swine herds of the Experiment Stations of the United States, Warwick[6] found that 1.73 per cent were herniated, of which over one-fourth of the males and nearly all of the females had the umbilical type of hernia. He concluded that umbilical and inguinal hernias occur to about the same extent in swine but are largely if not entirely independent in their incidence. On this basis it is estimated that there occurs annually in the swine herds of the United States alone approximately 150,000 cases of umbilical hernia in males and 230,000 cases in females.

Warren and Atkeson[5] reported the occurrence of umbilical hernia in one female and 20 male calves of a family of Holstein-Friesian cattle. All could be traced to one animal. On the basis of their data they judged the defect to be sex-limited, being a dominant character in males but in females the mode of inheritance was questionable. Geiger, Steenbock and Parsons[1] in a study of lathyrism in rats incidentally observed umbilical hernias in 10 rats, or 25 per cent of the young animals, receiving a ration containing 50 per cent of sweet peas. Stockman[4] in his comprehensive review of lathyrism, however, does not mention hernia in connection with this condition. Umbilical hernia in other species of animals and man is regarded by some writers as hereditary, but no studies involving significant numbers have been made as with inguinal hernia.[3]

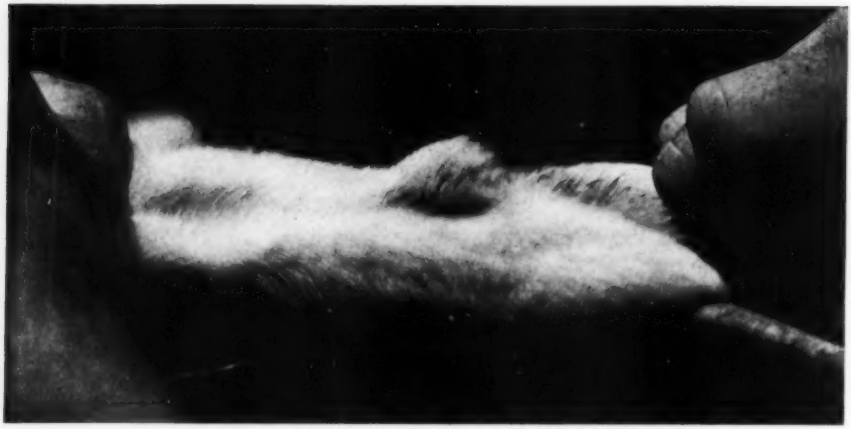
Several years ago umbilical hernias were observed in a litter of our experimental colony of white rats. Realizing the need for information concerning the possibility of transmission of this defect and appreciating the importance of a small animal in securing adequate data, it was decided to investigate this problem.

Description of Umbilical Hernia

Umbilical hernia is a protrusion of abdominal contents through the umbilical ring—an opening in the abdominal wall through which the umbilical cord had passed in fetal life and which normally closes shortly after birth. If there is a failure in proper closure, the skin covering this region cannot withstand intra-abdominal pressure with the result that a hernial sac is formed (Figure 10). This is composed of the peritoneum inclosing the protruding intestines as well as tissue layers outside it in this area. Growth may cause an enlargement of the hernial ring as well as a distortion of the elements that are contained in the hernial sac. In one animal the defect was of such an extent that it involved most of the abdomen. The accompanying histological photo-micrographs (Figure 9) of two moderately severe cases show that the peritoneum had protruded through the hernial ring.

In swine, umbilical hernias are usually first noticed after the animals are several months of age. In the present work the existence or non existence of hernia

*Published with the permission of the Director of the Experiment Station as Journal Article No. 256 (n.s.). The authors wish to express their appreciation to Professor H. R. Hunt of the Zoology Department for his kind advice and interest in this problem and to Professor E. T. Hallman of the Pathology Department for the use of laboratory facilities in preparing slides of the hernia.



EXTERNAL APPEARANCE OF HERNIA

Figure 10

The umbilical hernias observed in this study varied in size from less than two millimeters to more than eight millimeters in diameter. A tendency toward hernia is undoubtedly hereditary, but simple mendelian inheritance was not established.

was determined at an age of 3 to 4 weeks so that the hernia observed must have been due to imperfect closure of the umbilical ring.

Experimental Procedure

Umbilical hernias in this study were first observed in all individuals of a litter consisting of one male and three females. The rats were of an albino strain obtained a number of years previously from a Columbia University colony. Except for the herniated condition, they appeared normal in all other respects. The three females of this litter were mated with the male, and hernia was found in their offspring. The concentration of the factor responsible for the development of hernia was then carried out by selecting females which had hernia and mating them with males which had the largest and most prominent hernias.

In order to obtain information as to the possible mode of inheritance, the herniated rats were also mated with animals which supposedly did not carry the hernia factor or factors as denoted by the absence of hernia. These normal animals were obtained from the Wistar Institute and from the University of Wisconsin. Several types of backcrosses were also carried out.

The females were mated in colony cages and when pregnant were placed in individual cages where the litters were born. At 3 to 4 weeks of age the abdomen in the region of the umbilicus was wetted with water with the help of a stiff brush so as to make the hair lie flat against the body in order to observe the presence or absence of hernia. Intra-abdominal pressure was increased by stretching the rat out and rubbing its back over a corrugated surface or by having a second operator pinch the skin with forceps so that the rat would struggle.

This procedure permitted observation of the presence or absence of umbilical hernia, and, if present, its size. An arbitrary scale of ½, 1, 2, 3, and 4 was used to grade the size of the hernias. On this scale "1" represented a hernia of approximately 2 millimeters in diameter while "4" represented a hernia of 8 or more millimeters in diameter. Hernias of size "½" were those that were perceptible yet less than 2 millimeters in diameter.

All animals were fed the regular

colony ration which was supplemented at various times with yeast, whole milk powder, extra cod liver oil, green alfalfa, and tomato juice to provide against the possibility of a deficiency in one of the recognized nutritive factors. It contained the following ingredients:

	Per cent
Ground yellow corn	36
Ground barley	30
Skim milk powder	20
Blood meal	5
Alfalfa leaf meal	5
Salt	1
Cod liver oil	1
Bone Meal	2

Analysis of this ration showed that it contained 18.2 per cent protein, 3.49 per cent fat, 3.93 per cent crude fiber, and 5.7 per cent ash.

In order to evaluate each individual in terms of the per cent of herniated "blood" carried, 25 per cent was allowed for hernia in each of the immediate parents, 6.25 per cent in each of the grandparents and 1.56 per cent in each of the great grandparents. The sum of these was considered the percentage of herniated "blood" carried by a particular individual. While it was realized that this method has its limitations it proved to be a valuable means of summarizing these particular data.

Results

Of 1,058 animals used in this study 414 or 39.1 per cent were herniated in some degree. The results of crosses important from a genetical point of view have been summarized in Table I, where the matings have been classified as to types of cross made. In this table the positive sign (+) means that the animal had an umbilical hernia while the negative sign (—) means that the animal did not have one. Where a negative sign occurs in the first generation, the animals were obtained from outside sources which were supposedly not related to the herniated stock. A positive sign in the first generation refers to a herniated individual of the original litter of one male and three females. Reading upwards gives the known ancestry of the individuals under consideration in the tables. For example, the second mating in Part *D* of Table I reads: $\begin{matrix}++ & -+ \\ + & - \end{matrix}$. This means that a herniated male, descended from mating a herniated male and female, was crossed on a non-herniated female descended from mating a non-herniated male with a herniated female. The following would be a diagrammatic expression of the above example:

$$\begin{matrix} + & + & - & + \\ (\text{Hern.}\,\delta)\times & (\text{Hern.}\,♀) & (\text{Nor.}\,\delta)\times & (\text{Hern.}\,♀) \end{matrix}$$

$$\begin{matrix} + & & + \\ (\text{Hern.}\,\delta) & \times & (\text{Hern.}\,♀) \end{matrix}$$

Mating the lower two animals produced the individuals under consideration in the table. The symbols used indicated phenotype only.

In order to conserve space, not all of the matings made have been placed in Table I. Only those which possessed the most value from a genetical point of view have been included.

Effect of Percentage of Herniated "Blood" on the Incidence of Hernia

It was recognized early in this work that umbilical hernia was transmitted frequently to rats of herniated ancestry. This is strikingly brought out in Table II where all the matings were grouped in 10 per cent herniated "blood" classes. Animals with hernia increased from 2.7 per cent where the parents had 10 to 19 per cent of herniated "blood" to 71.2 per cent where the parents had 80-89 per cent of herniated "blood." The rather high value 17.6 per cent in the 0-9 per cent class is due to small numbers and the presence of hernia in one litter of which the presumably normal male was obtained from a colony which we learned later had had an introduction of herniated stock.

Effect of Selection and Inbreeding

When the original male was mated with the three original females, 51

progeny were obtained of which 43.1 per cent were herniated, as shown in the first line of Part *A* of Table I. When second generation females were mated with the original male, 61 young were produced, of which 78.7 per cent were herniated, as shown in the second line. When males and females were both of the second generation, 31 young were produced of which 61.3 per cent were herniated, as shown in the fourth line. When second generation males were mated with a third generation female, 9 young were produced, of which 55.6 per cent were herniated as shown in the fifth line. When males and females of the third and fourth generations were mated with each other, 49 young resulted, of which 71 per cent were herniated. Not all the data for all the latter matings are shown in Table I.

Effect of Out-Crosses on Occurrence

When animals with a history of hernia were mated with normal animals varying results were obtained. This was true whether a herniated male was crossed with a normal female or the reciprocal cross was made. All five normal males used, supposedly unrelated to the herniated animals, produced herniated progeny when mated with herniated females. One male obtained from the Wistar Institute produced only three individuals with hernias of size 1 out of a total of 59 young, when crossed on herniated females. Where the hernias were size 1 there could be little doubt of their actual existence. Another male produced one hernia of size 2 out of 22 young. When the 5 males without history of hernia were mated with herniated females, 20 of a total of 112 young were herniated, or about 18% (see part *B* of Table I).

When 18 females of negative history in so far as hernia was concerned were mated with males with hernias, 137 young were produced of which 25, or 18 per cent, were herniated as shown in Part *C* of Table I. The 25 hernias were from 11 different females. Some of the females were mated only once and many had a relatively small number of young in their litters, so one cannot say that the remaining 7 females would not have produced herniated young if rebred a number of times. Of a total of 249 young produced by the above two reciprocal matings, 45 animals or about 18% were herniated.

It is evident in Part *C*, Table I that when the original male was crossed on non-herniated females only 7 per cent of hernias were produced while when inbred second and third generation males were used on similar females the incidence of hernia was 18 to 38 per cent respectively. This holds for the reciprocal of these crosses for the first and second generation females but not for the third generation as is shown in the first 3 crosses in Part *B* of Table I. Here, however, only 15 animals are involved, which includes only 3 litters produced by the male obtained from Wistar Institute.

Effect of Back-Crosses

In order to get some indication of the possible mode of inheritance normal males were mated with herniated females and the F_1 generation females which did not possess hernias were mated back to herniated males. Of 72 young produced in this manner, 18 or 25 per cent were herniated as shown in Part *D* of Table I.

When herniated males were mated with normal females and the F_1 generation females which did not possess hernias were back-crossed with herniated males, 174 young were produced of which 41 were herniated or about 22 per cent as shown in Part *E*.

When a herniated male was mated with a normal female and the F_1 generation non-herniated females mated back to a normal male, 3 out of 17 young produced were herniated, or about 18 per cent as shown in Part *F*. There are hardly sufficient numbers of offspring in this particular type of mating to be of significance.

When males and females both possessing hernias were mated, and the F_1 female progeny, which did not possess hernias were mated back to herniated

males, 29 of the 54 progeny produced or 54 per cent, were herniated as shown in Part *G*.

Occurrence with Relation to Sex

Of 414 herniated rats 185 or 44.7±1.65 per cent were males and 229 or 55.3±1.65 per cent were females. The incidence of sexes should be equal so that the probable error of the difference, 5.3, being 1.65, indicates that the difference is on the border line of significance. Where herniated rats were mated with normal rats, as shown in Parts *B* and *C* of Table I, 127 or 47.2±2.05 per cent of 269 individuals were males, which is not statistically significant. Considering that there should have been 142 males instead of 127 there was a deficiency of 10.6 per cent males.

When individuals which possessed herniated "blood" were mated together 789 progeny were produced of which 341 or 43.0±1.18 per cent were males and 448 or 57.0±1.18 per cent were females, which difference is statistically significant. Since there should have been the same number of males as females or 448 there was a deficiency of 24 per cent of males.

Effect of Percentage of Herniated "Blood" on Size of Hernia

In correlating the percentage of herniated "blood" present in the parents with the size of hernia in the offspring there was a positive correlation of 0.15 ±0.03, which is rather low. By referring to Table II it will be seen that a greater number of larger sized hernias appeared where the parents possessed a higher percentage of herniated "blood."

Variation in Hernias in Different Litters from Identical Matings

Some variation occurred where identical matings were made several times in succession, as shown in Table III. This variation, however, was probably no greater than would be expected, except in the progeny of male 50 and female 29. In the latter case no hernias were produced in the first 4 litters, containing a total of 19 young, while the fifth mating produced 5 herniated animals in a litter of 10.

Discussion

It is quite evident from the data presented in Table II that as the percentage of herniated "blood" increased, the incidence of hernia also increased, since the percentage of herniated offspring increased from 2.7 per cent in the 10-19 per cent herniated "blood" group up to 71.2 per cent where the parents possessed 80-89 per cent of herniated "blood."

The effect of selection and inbreeding was shown to have a pronounced effect on the incidence of hernia, since the incidence was materially increased from 43.1 per cent herniated as a result of crossing the original litter mates together, up to as high as 78.7 per cent in some of the succeeding generations. This incidence is considerably above that obtained by Warren and Atkeson[5] for umbilical hernia in calves and by Warwick[6] for inguinal hernia in swine. The latter worker obtained an increase in the percentage of inguinal hernias from 14 to 42 and 43 per cent in the second and third generations, respectively, by controlled matings.

When normal animals were mated with animals with a history of hernia, approximately 18 per cent of the young produced were herniated. There seemed to be considerable variation in the number of hernias which each of the 5 normal males produced in this type of mating. However, since the males used were not mated to the same females and since identical matings show some variation, the difference in the ability of the normal males to produce herniated offspring is probably not significant.

Where certain types of back-crosses were made, little information as to the mode of inheritance was obtained except that phenotypically normal animals which have descended from herniated individuals possess considerable ability to transmit the hernia character to their offspring if mated with an individual which possesses genes for hernia.

The results of these back-crosses also indicate that both sexes possess approximately the same ability to transmit the character under similar conditions. In reciprocal out-crosses practically the same average percentage of hernias was obtained, as is shown in Parts *B* and *C* of Table I. Further evidence of this is deduced from the fact that when a normal male was crossed with a herniated female and the non-herniated females back-crossed to a herniated male, as shown in Part *D*, practically the same number of hernias were obtained as when a herniated male was crossed with a normal female and the non-herniated females back-crossed with a herniated male, as shown in Part *E*.

Evidence that the hernia is not sex-limited is shown by the fact that out of a total of 414 herniated individuals, 185 or 44.7 per cent were males and 229 or 55.3 per cent were females. In swine the inguinal type of hernia was found only in males, as shown by Warwick.[6] Warren and Atkeson[5] found only one herniated female in their study of umbilical hernia in calves and concluded that umbilical hernia in calves was sex-limited.

There was found an apparently significant difference between the number of males and females from parents containing herniated "blood." This indicated a deficiency in the number of males. It is unknown whether the mortality of males occurred during gestation or during the first few weeks of life. Therefore no explanation is offered for this apparent difference.

A small correlation (+0.138) was obtained between the percentage of herniated "blood" present in the parents and the size of hernia in the offspring. It would seem that this correlation would increase with further selection and inbreeding and with more knowledge of the genotype of the individuals.

There was no significant correlation (—0.0008) between the size of hernia of the parents with that of the offspring.

The probable errors of the difference between the percentages of herniated animals produced in the various classes of Table I were computed. There were no significant differences between groups *B* and *C*, *B* and *D*, *C* and *D*, and *G* and *A*. There were significant differences between *B* and *G*, *B* and *A*, *C* and *G*, *C* and *A*, *D* and *G*, and *D* and *A*.

Table I. Number, Size, and Distribution of Umbilical Hernias in Certain Selected Matings

♂	♀	Males Herniated No.	Males Normal No.	Females Herniated No.	Females Normal No.	Total No.	Total Per cent	Herniated "Blood"	Size of Hernia 1/2	1	2	3	4
B	+	10	14	10	15	49	40 45.1	20.0	5	10	3	3	1
	+	21	6	27	7	13	48 78.7	41.0	11	13	15	3	6
	+	0	1	1	0	1	1 20.0	40.0	1	0	0	0	0
	+	9	6	10	6	12	19 61.3	72.0	10	7	1	0	1
	+	6	2	3	2	4	9 69.2	70.1	1	4	4	0	0
	+	13	9	11	9	5	24 65.8	61.2	8	9	6	1	0
		59	31	63	37	66	120 61.3	47.19					
B	+	0	6	1	13	21	1 4.5	25.0	0	0	1	0	0
	+	9	11	6	11	22	11 33.3	27.5	8	2	0	0	1
	+	0	9	0	6	15	6 0	40.0	0	0	0	0	0
	+	2	3	4	2	5	6 64.6	32.0	1	3	0	0	0
	+	0	2	0	4	6	0 0	34.4	0	0	0	0	0
	+	0	8	0	7	23	2 11.8	23.0	0	2	0	0	0
	+	2	2	0	2	2	2 0	44.1	0	0	1	0	1
		13	41	11	45	94	28 17.2	31.84					
C	+	9	39	1	39	76	5 7.4	28.0	4	1	1	0	0
	+	1	2	1	7	9	2 10.2	27.5	2	0	0	0	0
	+	4	12	11	14	25	12 27.6	40.0	10	1	0	0	0
		14	53	13	60	110	19 14.36	32	16	2	1	0	0
D	+	5	11	3	14	25	8 24.2	31.2	3	3	2	0	0
	+	4	10	3	12	22	7 23.0	42.7	2	1	0	0	0
	+	0	1	1	4	7	1 12.5	20.0	1	0	0	0	0
		9	22	7	30	54	16 22.22	31	6	4	2	0	0
E	+	6	20	12	20	62	18 17.0	31.2	9	5	4	0	0
	+	10	20	11	17	28	22 21.7	45.7	7	11	4	1	0
		16	40	23	37	130	40 20.41	13	16	5	1	0	
F	—	1	10	2	4	14	3 17.64	27	1	2	0	0	0
G	+	2	1	4	2	5	6 66.7	27.5	0	2	2	2	0
	+	3	2	10	10	20	20	20.0	13	5	4	0	0
		5	10	14	12	25	26 34.16	15	5	4	2	0	

Table II. Relation of Number, Size, and Distribution of Umbilical Hernias in White Rats to the Percentage of Herniated "Blood"

Herniated "Blood"	Normal No.	Herniated No.	Herniated Per cent	Size of Hernia 1/2	1	2	3	4	Size of Hernia 1/2	1	2	3	4
									Per cent				
0 - 9	16	2	17.6	1	1				5.9	11.8			
10 - 19	36	1	2.7	1					2.7				
20 - 29	99	7	6.6	4	1	1			3.9	1.0	1.9		
30 - 39	170	54	25.5	24	19	8	2	1	10.4	8.2	3.4	0.9	0.4
40 - 49	155	57	27.1	20	17	4	1		10.6	8.1	1.9	0.5	
50 - 59	31	60	66.9	18	16	7	3	1	10.8	16.7	7.3	2.1	1.0
60 - 69	19	50	72.6	12	15	14	3	6	17.4	21.7	20.3	4.5	8.7
70 - 79	99	103	63.6	63	26	13	1	4	38.6	15.7	7.9	0.6	2.5
80 - 89	38	94	71.7	34	34	17	7	2	25.7	25.8	12.9	5.3	1.5
90 - 99	0	0											

Table III. Number and Size of Umbilical Hernias in the Progeny of the Same Mating at Different Times

Male No.	Female No.	Litter	Males Herniated	Males Normal	Females Herniated	Females Normal	Size of Hernia Normal	1/2	1	2	3	4
50	1	8-23-22	1	3	1	3	6		1			1
		10-13-22	1	0	0	3	3					
		1-8-23	0	2	1	3	5			1		
		2-10-23	0	0	0	1	1		1		1	
		5-20-23	0	1	0	3	3	1				
93	5	7-27-22	0	1	1	0	2	0	1			
		11-10-22	0	0	0	0	0		1		1	
		1-17-23	1	0	0	1	1					
17	18	1-20-23	0	0	0	1	1	0	1			
		2-17-23	0	1	0	1	1	1				
		5-21-23	0	0	0	0	0		1		1	
		8-2-23	1	0	0	1	1					
60	65	11-20-22	0	3	0	3	6					
		1-20-23	0	2	0	0	2					
		7-28-23	0	1	0	1	2					
		9-10-23	0	0	0	0	0	4	1			

The out-crosses indicate that the character may be recessive, but not a simple recessive, if the assumption is made that the character is wide spread. Such an assumption would be tenable since the occurrence of umbilical hernia might easily pass unnoticed in most laboratories. Incidentally the authors are aware that the defect has been noticed in two other widely separated laboratories. On this supposition it seems very probable that homozygous normal individuals were not secured for making these out-crosses since about 18 per cent of the F_1 generation were herniated. The normal females used would appear to be of almost the same genotype as the females produced from mating herniated with normal animals, since in the back-cross they produced 23 per cent hernias, about the same as that produced by crossing herniated males with the supposedly unrelated normal females. However, the suggested recessive nature of the character cannot be accepted as fact, since it is based on an assumption that homozygous normal animals were not obtained. The data do not permit a simple tenable theory of the exact mode of inheritance of this character. When homozygous animals are obtained, both normal and herniated, more significant results will be obtained from the genetic viewpoint.

In the present work the effect of various modifying factors such as the effect of ration and environment, was not determined.

It was noticed during the latter part of the investigation that the size of litters had decreased considerably and that frequently the young did not suckle properly, with the result that they died during the first day or so of life. Those that lived seemed to grow rather slowly and never attained a very large size on a ration which has been satisfactory for another colony of rats. This unthriftiness was possibly due to the fact that the mothers had had a number of litters without proper rest periods and had become older than desirable for breeding purposes. Whether this unthriftiness had any influence upon the results obtained in this study is unknown.

It might possibly be considered that umbilical hernia typifies failure in normal closure. There are other fairly common instances of imperfect closure of an embryological origin such as cleft palate, harelip, and patent foramen ovale, which are believed to be inherited, but are difficult to investigate due to lack of sufficient numbers and the lethal nature of some of these tendencies. However, the similarity of the underlying causation of these defects to umbilical hernia and their possible hereditary nature are pointed out.

Certain practical applications seem to be justified on the basis of the work reported. In livestock breeding the male is more important than the female since it is usually the parent of more animals than the female. Since one herniated parent can throw a considerable number of umbilical hernias even when the other parent is normal, particular care in the selection of males free from hernia is indicated. If umbilical hernias appear in the breeding stock it would seem proper to reject from the breeding pen siblings of the affected animals as well as the parent or parents responsible for transmitting the defect. In time this would prevent development of herniated progeny from the matings of normal animals of certain genotypes.

Summary

Controlled matings of white rats having umbilical hernia have been made.

The data on 414 herniated individuals of a total of 1,058 animals clearly show that umbilical hernia is an inherited character.

By selective inbreeding it was possible to increase the percentage of umbilical hernias in litters to approximately 70 per cent. By similar means the size of the hernia was increased.

The influence of the male and female are of about equal importance in its transmission.

The data indicate that umbilical her-

nia may be either a blended or multiple factor character. It is not sex-limited.

Of 789 individuals from parents containing herniated "blood", 341 or 43.0 ± 1.18 per cent were males—an apparent significant deficiency of males.

The effect of modifying factors such as ration was not determined.

Practical procedures are considered for the eradication of umbilical hernia from breeding stock of the different classes of farm animals.

Certain analogous defects of apparently similar causation are discussed.

Literature Cited

1. Geiger, B. J., H. Steenbock, and H. T. Parsons. 1933. Lathyrism in the Rat. *Journal of Nutrition*, 6:427-442.

2. Hadley, F. B., and B. L. Warwick. 1927. Inherited Defects of Livestock. *J. of Amer. Vet. Med. Assoc.*, 70, new series, 23:492-504.

3. Moorhead, J. J. 1932. Traumatic Inguinal Hernia. *J. of Amer. Med. Assoc.*, 98:1785-1788.

4. Stockman, R. 1931. The Poisonous Principle of Lathyrus and Some Other Leguminous Seeds. *Jour. Hygiene*, 31:550-562.

5. Warren, T. R., and F. W. Atkeson. 1931. Inheritance of Hernia. *Jour. of Hered.*, 22:345-352.

6. Warwick, B. L. 1926. A Study of Hernia in Swine. *Wisconsin Agr'l Expt. Sta. Research Bulletin*, 69:1-26.

◆

THE JEW IN SCIENCE

THE purpose of this volume* is "to direct the attention of the Jew and non-Jew to the accomplishments by Jews in scientific endeavors, so that Jews may know themselves better, and that non-Jews, where [an antagonistic attitude] is the product of ignorance rather than malice, may avoid making prejudiced statements." The work is divided almost equally into a section on the history of Jewish contributions to science from earliest times, with classified lists of outstanding contemporary Jewish workers in various scientific fields, both theoretical and applied. There is a brief chapter tracing the history of the dispersion of the Jews and the recurrent waves of persecution to which they have been subjected. The author avoids the elementary error of regarding the Jews as a biologically racial entity, but many of his statements imply equally questionable assumptions concerning the cultural or "spiritual" homogeneity of the Jewish people.

Obviously, the timeliness of this volume depends on the tacit admission that anti-semitism, already crystallized as an official philosophy in Germany, is showing alarming signs of recrudescense in other parts of the world, not excluding the United States. It appears to the reviewer that the author would have performed a much more valuable work if he had made an effort to discover the factors behind this recrudescence. Many of the conditions which Kautsky described ("Are the Jews a Race," English translation, 1926) as leading to hostility against the Jews in the past history of anti-semitism seem strikingly analogous to the conditions which have been associated with current anti-semitism. A recognition of these conditions is clearly a prerequisite to their correction, and an analysis of the growth of anti-semitism in relation to the changing social, political, and economic milieu would be a first step in this direction.

A notable defect of the book is the ineptitude of its style, which is wholly incommensurate with the considerable labor which must have gone into the compilation of the material.

Paul R. David
Storrs Agricultural Experiment Station.

*THE JEW IN SCIENCE, by Louis Gershenfeld. Pp. vii + 224. Philadelphia: The Jewish Publication Society of America, 1934.

"HEREDITY" LANTERN SLIDES

MAN

16-397. Gregor Mendel's Handwriting — First Page of "Studies of Plant Hybrids".
17-435. Merton House—Home of William Bateson.
21-1-Cover. The "Fuchsia-Picture" of Mendel.
21-No. 2-Cover. Francis Galton.
26-158. Ishmael Family—Six Generations of a Degenerate Family.
26-159. Roosevelt Family—Pedigree Chart of a Superior Family.
26-292. Dr. and Mrs. Hugo de Vries.
15-330. Fused Finger Joints.
17-393. Short Legs—Inherited Achondroplasia.
20-75. Extra Fingers—Recessive Polydactylism.
20-74. Chart Showing Inheritance of Polydactylism.
20-352. A Folding Man — Hereditary Absence of Clavicles.
22-104. Crooked Arms and Legs—Hereditary Bone Fragility.
23-307. Five Generations of Glaucoma.
23-425. Inherited Doublejointedness in the Thumb.
24-269. Chart Illustrating Sex-Determination.
22-47. Palm Prints of Fraternal Twins.
22-40. Corresponding Fingerprints of 3 Pairs of Identical Twins.
23-369. Identical Twins—Separated 13 Years.
23-2. Identical Twins Reared Apart—Mary and Mabel when Grown.
23-5. Identical Twins Reared Apart—Mary and Mabel as Babies.
23-6. Identical Twins (Mary and Mabel) at Two Years—Reared Apart.
23-8. Palm Prints of an Identical Twin (Mary).
23-9. Palm Prints of an Identical Twin (Mabel).
24-443. Mirror-Imaging in Eye Dominance in Identical Twins.
24-444. Table Classifying Handedness in Twins.
25-140. Table of Physical Characteristics of Identical Twins Reared Apart.
25-141. Identical Twins (Harold and Holden) Reared Apart.
25-293. Retina of Normal and Albino Eyes.
25-332. Abnormal Fingers and Toes — Syndactyly, Camptodactyly, and "Hammer Toe".
25-333. Short Finger (Brachymetapody) due to short metacarpal bone.
25-391. Hereditary Absence of Incisors.
25-446. Inherited Chest Depression—Koilosternia.
25-483. Sex-Linked Toothlessness.
25-338. Fifty-fifty Segregation for Woolly Hair Mutation.
26-143. Extra Toes—Inherited Polydactylism.
10-389. Twin Acrobats—Identical Twins.
10-409. Typical Identical Twins.
20-50. Identical Twins Reared Apart.
20-52. Palm Patterns of Identical Twins.
20-155. City Boy and Country Boy—Identical Twins Reared Apart.
20-160. Enlarged Prints of Left Ring Finger of Identical Twins (C & O).
22-202. The Hilton Siamese Twins.

ANIMAL

16-154. Pelvic Bones of Normal and Rumpless Fowls.
16-352. Leg Bones of "Reptilian Fowl" and of Normal.
19-413. A Fertile Mare Mule.
19-414. "Old Beck" (Mule) and Her Horse-Like Colt.
19-415. Mule-Like Colt of Fertile Mare Mule.
20-112. Recessive Leg Feathering in Fowl.
20-113. "Heel Tufts" in Fowl.
21-323. Heterogeneous Chromosome Structure — Irregular Cell Division in Drosophila Melanogaster.
22-213. Conjoined Twins in Cattle.
22-296. Backcrosses between Hybrid (Zebu × Yak) Female × Zebu Male.
22-298. Zebu Bull Used in Yak Crosses.
22-299. Yak Cow Used in Zebu Crosses.
22-305. A Zebu-Yak Hybrid—4 Years Old.
22-305. Zebu-Yak Hybrid Cow—4 Years Old.
23-449. A "Flightless" Rooster.
24-183. Parallel Color Mutations (Himalayan and Siamese) in Rabbit and Cat.
25-208. Five Generations of Inbreeding—Shorthorn Cattle (chart).
25-266. X-Ray Photos of Normal and "Flightless" Feathers in Fowl.
25-435. Production of Agglutinins in Rabbits (chart).
25-432. Agglutination of Incompatible Bloods.
25-464. "Portraits" of Salivary Gland Chromosome.
25-466. Giant Chromosomes Compared with "Normal".
25-469. Synapsis of Normal and Inverted Chromosomes.
25-470. Synapsis of Deleted and Normal Chromosomes.
25-472. Cyto-Genetic Maps of Salivary Chromosomes. (Insert.)
26-62. Salivary Chromosomes and Gonial Chromosomes Compared.
26-62. Reference Map of the Salivary Chromosomes of Drosophila. (Insert.)
26-182. Structure of Salivary Gland Chromosome.
26-301. Effects of Sex Hormone Injection on Feather Growth.

PLANT

15-204. Capsules of Globe Daturas.
17-372. Awn of Manchuria (Rough) Barley.
17-373. Awn of Lion (Smooth) Barley.
18-128. Adaptive Cupped Tendrils of a Plant.
18-129. Adaptive "Vacuum Cup" Toes of a Toad.
18-382. Leaves of Live Oak × Post Oak Hybrids.
18-383. Leaves of Overcup × Live Oak Hybrids.
19-419. "Fatback" and "Razorback" Honey Locust Pods.
20-24. Haploid and Dwarf Diploid Tomato Plants.
20-25. Haploid and Normal Tomato Plants.
20-26. Haploid and Diploid Tomatoes.
20-206. Flax Hybrid Produced by Artificial Nutrition.
22-368, 370. Plant Patents No. 2 and No. 3.
24-131. Radium-Induced "Ridged" Tomato.
24-133. Chimeral Radium-Treated Tomato Leaves.
24-134. Radium-Induced Virescent-White Tomato Seedlings.
24-368. Gene Mutation Chimera in Delphinium.
26-137. Ears of Maize, Relatives, and Hybrids.
25-448. Fasciated Sugar Beet.

Above are listed ninety Lantern Slides from JOURNAL OF HEREDITY illustrations. These are only a sample of over five thousand charts and photographs which have been published in the twenty-six volumes of the JOURNAL. Order from this list by Number. Slides may be ordered for any illustration appearing in the JOURNAL by giving Volume—Page—and Figure Number. Slides on this list will be supplied within a week of receipt of order. Price 75c each —$8.00 per dozen. Slides are numbered according to volume and page on which they appear. Thus 25-372 is from Volume 25, page 372 of the JOURNAL, 24-300 is from Volume 24, page 300, etc. Suggestions for illustrative material from the JOURNAL suitable for specific lectures will gladly be furnished on request. Address orders and inquiries to:

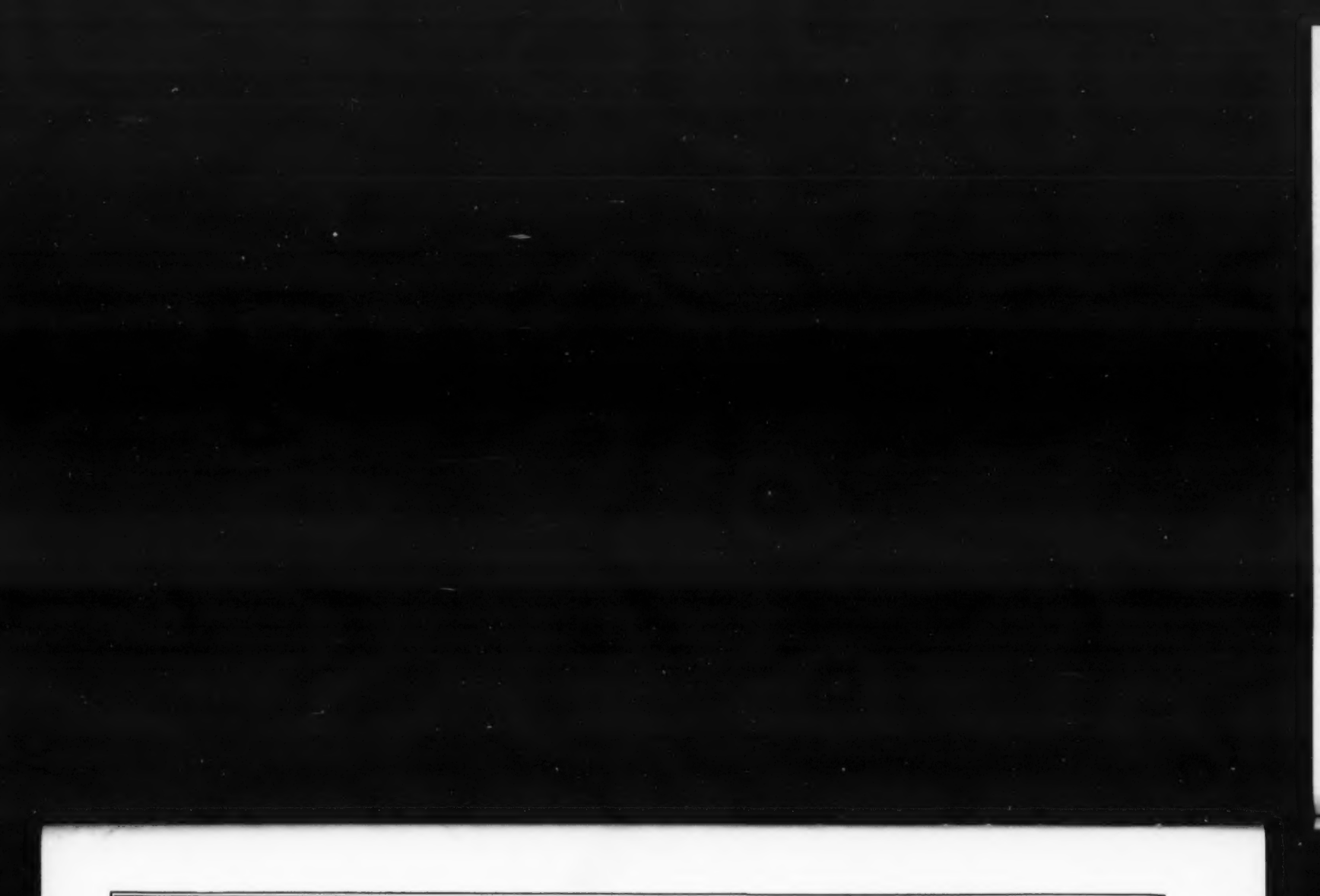
AMERICAN GENETIC ASSOCIATION

Victor Building **Washington, D. C.**

THE PUBLICATION BONUS

WHETHER one copy of the JOURNAL is printed or ten thousand, many of the larger costs of preparation would be the same. The entire expense of typesetting, halftone engraving, editing, correspondence with authors regarding articles, proof, etc., must be incurred before a single copy can be mailed. On the most economical basis these preliminary expenses would amount to over $400.00. If ten thousand copies are printed instead of one, the additional cost of paper, of binding and trimming the extra copies, etc., would amount to only about $400.00. Thus, the cost of the 9,999 additional copies would be less than that *first* expensive copy. The cost of these additional 9,999 copies is about *four cents each.* If we lump our expenses for these two runs, and distribute all the costs over ten thousand copies, the production cost of each copy is eight cents, as compared with a production cost of $400.00 for publishing one copy.

These facts have an interest to every member of the A. G. A. because they have a very important bearing on the future of the JOURNAL—economic conditions being as they are. The greater part of the income available for publishing the JOURNAL is derived from the dues paid by every member. Whenever a member withdraws for economic or other reasons, our income is reduced $3.00 *but our expenditures cannot be reduced in proportion.* It is easy to see that even a rather small percentage of withdrawals may threaten with financial disaster a publication operating under the margin between available funds and necessary costs. Thus a ten per cent reduction in membership and subscriptions (350) means about $1,100 less income, but our production costs can be reduced only about $175.00, leaving a deficit of nearly a thousand dollars to be met in other ways! * * * The publication of the JOURNAL is made possible by these individual contributions which, "though but a little to each of you, represent a goodly sum in the aggregate."

THE above statement regarding the paradox of the printing press was published in the JOURNAL shortly after the bank holiday in 1933, when finances were at their lowest ebb. If conditions had stayed as they were then, the JOURNAL would long since have ceased to appear. The improvement has been encouraging, but the paradox still dogs us, and will until the membership support of the JOURNAL exceeds 5,000. When that point is reached two things begin to happen. The law of diminishing costs sets in with considerable of a vengeance, and the JOURNAL begins to have much better possibilities as an advertising medium than it does at present. This bonus of operating economy and an advertising income will not be delivered on any one day by the postman, but many will share in it, for it will put the JOURNAL's financing definitely in balance, and it will assure a better publication.

The JOURNAL has served a unique and a useful purpose. The articles it has been privileged to publish, and its unique file of illustrations are widely appreciated and used in many ways. In view of this situation it is obvious that all those who realize the value of the JOURNAL and appreciate the importance of what it has done in furthering genetic enlightenment should support it directly to the extent of subscription or membership. Reading the JOURNAL in the library, and borrowing a copy from a member does not help us to resolve the printer's paradox. If you read the JOURNAL and are not a member, join today. Checks, stamps, money orders, and 59-cent dollars are all acceptable. Let's beat the printing press at its own game and cash in on the Bonus of diminishing costs!